VOL. XIX. No. 4.

ARCHIVES OF OTOLOGY.

REDUPLICATIONS OF MUCOUS MEMBRANE IN THE NORMAL TYMPANIC CAVITY—THEIR CLINICAL IMPORTANCE.

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An important factor in the persistence of chronic suppurative disease of the middle ear, and of the effects which that and the various chronic non-suppurative diseases have subsequently upon the hearing by impairing the mobility of the ossicular chain and its motor complement, the membrane of the round window, is the presence of folds, bands, or reduplications of mucous membrane in the upper and posterior portion of the tympanic cavity. These have been studied and described by many observers, Morgagni, Toynbee, Hinton, Politzer, Kessel, Zaufal, Prussak, Gruber, Urbantschitsch, Miot, Gellé, and Gradenigo among them, and their possible clinical importance considered, but it is only recently that they have been the subjects of renewed interest in the light of other physiological and clinical contributions to our knowledge of the distribution and control of the circulation in, and the inception and course of diseases of, the upper portion of the tympanic cavity. In this connection it seemed worth while, when reviewing a series of examinations of temporal bones, which I had made fifteen or more years ago in reference to this point, to repeat the examination of a number of middle ears with greater care, bearing in mind the intermediate experience of others, which had confirmed the classification of these folds as to location, into those, horizontal, which had been considered by the clinician principally as obstructive to the drainage of the tympanic attic, and those, vertical or irregularly disposed about the stapes and round window, which had a clinical importance on account of their subse-

quent interference with the mobility of these parts, the third class being those of striæ and reduplications—principally the former—in the neighborhood of the mastoid antrum. The following descriptions are of selected cases, illustrating fully these first and second classes, the two of most frequent importance clinically. In both of the specimens it was evident that the middle ear had not been the seat of disease, and that the reduplications found were neither pathological in themselves nor rendered unduly prominent by any pathological process; they represent conditions which exist normally, to a greater or less degree, in more than 70 per cent. of human middle ears, and are therefore worthy of consideration from a practical point of view.

In the first case, the mastoid cells, antrum, and tympanic attic were large and well developed, the mastoid antrum was crossed by numerous striæ, and there was, in addition, a reduplication of the mucous membrane, triangular in shape, with its apex attached to the superior border of the short process of the incus, and its base, two millimetres in width, attached by striæ to the inner, posterior and outer walls of the mastoid antrum. There existed also the following reduplications of the mucous membrane, one from the anterior wall, at a height of about one millimetre above the level of the tendon of the tensor tympani, extending downward and backward to the tendon, and completely dividing the upper from the lower portion of the tympanic cavity anteriorly; above this, a second and smaller reduplication, extending from the base of the capitulum mallei downward to the tendon, and then forward to the centre of the first reduplication; thirdly, a small triangular fold, extending from the centre of the tendon along its border and the inner tympanic wall to the anterior crus and head of the stapes; and, finally, a firm reduplication, extending completely across the posterior portion of the cavity, and attached to and bounded by the stapes, descending process of the incus, lower border of the posterior pocket and posterior and inner walls of the tympanum, the upper portion of the cavity being separated from the lower completely, with

exception of an opening about one millimetre in diameter, between the descending process of the incus and the malleus.

In the second specimen the horizontal reduplications were few in number and comparatively insignificant. The stapes had a fold passing from one crus to the other and closing the space between with exception of a small spot at the apex of the triangular interspace; a second reduplication completely filled the space bounded by the eminentia stapedii, the tendon of the muscle, the posterior wall of the oval niche, and the posterior crus of the stapes; there were in addition numerous striæ and a narrow fold anteriorly, the latter at the bottom of the niche and extending from the anterior crus to the corresponding niche wall, and a small reduplication from the head of the stapes at the insertion of the tendon extending upward and backward, the plane of its surface corresponding to that of the tympanic wall.

The mucous folds in both these typically illustrative cases differed in clinical importance because of their structure as well as according to their position; the presence of fibrous tissue in a greater or less degree affecting the resistance of the mucous reduplications and sometimes placing them on the plane, mechanically speaking, of ligaments.

The first case corresponds very nearly to the one depicted in Dr. Bryant's plate, Fig. 11, and is an extreme example; the second is paralleled by cases included in his paper.[1]

Aside from the obstruction to free drainage of the upper portion of the tympanum, in the event of fluid exudation therein, the reduplications of mucous membrane of the first class may be considered as playing an important rôle in the etiology of those diseases of the tympanic attic the majority of which start with a suspense of vasomotor inhibition in that region; for, taking into consideration the fact that these folds are really folds or reduplications, and that each presents a double mucous surface (with or without, usually

[1] "Observations on the Topography of the Normal Human Tympanum"; the investigations on which Dr. Bryant's communication is based were begun with a view to mutual use and publication, but the independence and originality of his work merit for it individual presentation; the two papers should, however, though separately written, be considered together.

with, connective tissue interposed), and that these folds curve to meet each other and the tympanic walls and structures to which they are attached, we can readily understand that the secreting and vascular surface of the tympanic attic may be easily doubled or trebled by their presence, a condition which serves to account for the rapid development of congestive disorders in the upper portion of the tympanum, and for the often excessive and copious serous exudation, without calling into question any contribution from the mastoid antrum. Indeed, the presence of folds of the third class, together with the known tendency to excessive engorgement and swelling of the mucous membrane in the antrum, favors a closure of that communication with the mastoid and effects the isolation of the middle ear, a series of conditions which cannot be found better illustrated than in the acute congestions of the tympanic attic of reflex origin, a typical case of the kind being one of those in which the congestion of the ear has followed some irritation of the corresponding side of the face, exposure to cold, for instance, followed shortly, sometimes within an hour, by severe pain in the ear and head, tinnitus, vertigo, and sense of fulness in the ear, the objective appearances being a transparent membrana tympani, very much distended, and congested in its upper portion and along the manubrium mallei; paracentesis of the more prominent inferior posterior quadrant of the drum-head giving vent to air, which escapes with a sharp hiss, and without relief from the pain, which first begins to decrease spontaneously with the subsequent appearance of serous discharge. The effects in this class of cases, and also in those having almost invariably a precedent history of nervous over-tire, which evidence themselves first with a sense of fulness in the ear, giving place rapidly to pain, soon becoming unendurable, and in which free incision along the superior border of the membrana tympani from the short process of the malleus backward, and deep enough to include the deeper layer of vessels going to form the inner manubrial plexus, is the only form of phlebotomy adequately efficient, cannot be accounted for on the supposition that we have to

deal only with the limited area of vascular tissue which would be afforded by the normal tympanic attic without the addition of the mucous folds. In the first of the cases cited it is evident that the sudden congestion of the mucous membrane so far restricts and decreases the lumen of the tympanum, and with such rapidity, as to compress the air within the remaining portion of the cavity, and with the removal of the air-pressure the serous exudation following, but with a sufficiently appreciable limit of time, shows that the compression of the air had been effected by the engorgement of the mucous membrane and not by the encroachment of fluid. In the second class of cases the bleeding, sometimes of twenty-four hours' duration, followed by copious serous discharge, long-continued, is evidence of a larger area of supply than could be afforded by the tympanic attic alone, or explainable easily on any other hypothesis than that of a reinforcement, as it were, of its vascular area. Following these cases a step farther, into the stage in which a suppurative process may have been established, the presence of the mucous folds of the first class, horizontal, in the attic, should be remembered not only because of their possible interference with free drainage, already referred to, but because, from their considerable thickening and the adhesions consequent upon the contact of their ulcerated surfaces, they present a considerable obstacle to the free movement of the ossicula after the suppurative process has ceased; while the effect of the thickening and adhesions of the folds of the second class, about the stapes and neighborbood of the fenestra rotunda, upon the mobility of these less ponderable elements of the sound-transmitting mechanism, and the value of surgical interference for their removal in effecting the mobilization of the stapes, are sufficiently recognized.[1] In cases of destruction or large perforation of the membrana tympani, with spontaneous evulsion of the incus, the considerable impairment in hearing,

[1] In the pathological tympana examined the thickening of the mucous folds was especially marked in cases of old suppurative otitis media, and the greatest interference with movement of the stapes was afforded by adhesions behind and below; subsequent operative experience has confirmed this observation.

due to the immobility of the stapes from the adhesions and thickening in these mucous folds, an immobilizing effect still further increased by the unrestricted action of the stapedius muscle, has been improved by the division, first of the tendon of the stapedius and then of these folds. In an illustrative case of this kind the patient had considerable impairment in hearing in both ears as the result of suppurative otitis media following scarlet-fever in childhood: the right ear was practically useless; there was destruction of the membrana tympani; the ossicula were in place, without evidences of carious process, and with the articulations apparently intact, so far as any suppurative invasion was concerned; the tip of the manubrium mallei was in contact with and adherent to the promontory; the mucous membrane of the tympanic cavity was slightly congested and covered with mucous secretion. The application of an artificial drum-head (absorbent cotton soaked in borated vaseline) had no appreciable effect in improving the hearing; under good illumination and with the proper antiseptic precautions, including the careful cleansing of the canal by means of cotton-tipped probes dipped in weak solution of bicarbonate of soda and then in corrosive sublimate and alcohol, a knife, double-edged, round-pointed, and curved on the flat, dipped just before using into a solution of boracic acid in alcohol,[1] was passed upward into the tympanic attic, both behind and in front of the malleus and carried from before backwards with a view to dividing the horizontal folds, the presence of which had been previously determined by means of a light wire probe; the tip of the manubrium was then cut away from the promontory by means of a lance-shaped angular knife (similar to that figured by Miot[2]), and the artificial drum-head was again tried but without success. The next step was the disarticulation of the incus and stapes and division of the stapedius muscle,[3] which gave some improvement, but it was not until,

[1] The deposition of the boracic acid upon the small knife-blade renders it much more easily seen after its introduction through the speculum.

[2] Compare Bezold: Exp. Untersuch. über d. Schallleitungs app. des menschl. Ohres, *Arch. f. Ohrenh.*, 1880, p. 38.

[3] "De la mobilisation de l'etrier," by Dr. C. Miot, Paris, 1890, p. 33.

in addition, free division of the mucous folds between the stapes and the walls of the niche had been made, that the hearing was sufficiently improved with the use of the artificial drum-head to make it of practical value. In this case the adhesions binding the stapes to the niche wall were most marked and strongest on the posterior crus; the contraction of the stapedius, permitted by the abnormal conditions in the remainder of the sound-transmitting mechanism, having favored this condition. As a further illustration of the importance of bearing in mind the possible influence of these folds of the second class on the mobility of the sound-transmitting parts, may be cited a case in which the mobilization of the stapes had but little appreciable effect until the division of a fold extending across the membrane of the round window released that mechanical secondary to the excursions of the stapes, and the improvement in hearing became manifest.

There is still a class of cases, already alluded to, in which the possible effects of thickening of these mucous folds should be remembered, namely, the chronic non-suppurative cases characterized objectively by a slowly progressive and finally considerable thickening of the tympanic mucous membrane and sub-mucous tissues, and subjectively by much impairment of hearing, proportionately persistent and distressing tinnitus aurium, and the train of other discomforts which lead the patient to seek any apparently justifiable means of relief—the puncture of the drum-head, or trepanation of the mastoid, of Sir Astley Cooper's time, or the more recently practised removal of the membrana tympani, malleus, and incus, as proposed in its entirety by Kessel, an operation which has for its ultimate purpose in point of fact, first, free access of the sound waves to the stapes, second, relief of that ossicle from the incubus of its immobile and immobilizing companions, and finally still further mobilization of the stapes after it has been freed from the remainder of the ossicular chain. In such an operation, in addition to the division of the recognized attachments of the ossicula, the influence of resistance to the removal of the malleus and incus afforded by the

thickened mucous folds should no more be forgotten than in the cases of suppurative otitis media, with caries of malleus or incus, in which the removal of these bones is a part of a simply justifiable surgical procedure, for although in the fixation of the ossicula in cases of chronic non-suppurative disease the effects of the morbid process upon the articulations and constant attachments of the bones are the important factors, the thickening of the less constant bands and folds (which play the part of ligaments often), though much less than in the suppurative cases and often very difficult to determine on examination, should be considered.

That the operation of excision of the membrana tympani and evulsion of the malleus and incus in any case of chronic non-suppurative disease of the middle ear is a necessary operation, until other measures have been sufficiently tested, should remain a question, especially in consideration of the relatively important part which obstructive mucous folds about the stapes are alone capable of playing in the immobilization of that ossicle; and that the purpose for which the malleus and incus are removed in non-suppurative cases, namely, to free the stapes from their immobilizing weight and rigidity, can be equally effected, when at all possible, by division of the incudo-stapedal articulation and tenotomy of the stapedius through an opening in the posterior portion of the membrana tympani sufficiently large to permit ready access to the stapes and round window.

OBSERVATIONS ON THE TOPOGRAPHY OF THE NORMAL HUMAN TYMPANUM.

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(With colored plate containing fifteen figures.)

THROUGH the kindness of Professor Dwight of the Harvard Medical School, I have been able, during the winters of 1888–89 and 1889–90, to examine a number of temporal bones, in pursuance of the suggestion of Professor Blake, with special reference to the reduplications of the mucous membrane lining the tympanic cavity. These reduplications were found to be much more abundant than was expected, for not one of the tympana examined was free from them, and in several instances they occupied the whole cavity of the tympanum above the ossicles.

In examining the tympanum for mucous reduplications, great care must be exercised lest in the dissection some of the folds should be destroyed before they could be noted. I adopted the habit of opening the tympanum from two points,—the mastoid antrum, and the floor of the tympanum—as the surest way of preserving the folds. The specimens were first hardened in alcohol, and afterwards washed with dilute glycerine to prevent damage to the delicate folds by drying during the process of dissection. In spite of all precaution, it was rarely possible to make a complete examination of any one tympanum.

The mucous folds are arranged in three groups: 1. Those of the mastoid antrum. Here there is usually a central longitudinal band, with radiating lines and trabeculæ.

2. Those attached to the malleus and incus. These radiate from the three long axes of the bones. 3. Those of the stapes and round window, and the neighboring tympanic wall. The mucous folds about the stapes join the crura together or connect the stapes with the walls of the pelvis ovalis. The mucous folds which are attached to the ossicula perform essentially the function of the ligaments, binding them together or to the walls of the tympanum; and for this reason they might be classified as ligaments. The anatomical elements are also the same as in the ligaments, varying only in the number of connective-tissue fibres enclosed by the mucous membrane. The essential differences between the mucous folds and the ligaments are merely strength and constancy, though sometimes the strongest so-called mucous fold is stronger than the weakest ligament. These two qualities seem to vary in the same ratio, the more constant folds being also the stronger. When the ligaments and mucous folds are considered in the same group, they represent all grades of strength, from the anterior ligament of the malleus to the most delicate fibre of mucous membrane invisible to the naked eye. Some regularity in the arrangement of the folds which have not been classed with the ligaments seems to exist, but their most striking feature is their constant variation. This fact probably accounts for their having received so little attention. No one of them can be found perfectly developed in every tympanum. It is very common to find a mucous fold in an imperfect condition, that is to say, less extensive than it often occurs. The usual place of a fold may be occupied by a few fibres, or by a less extensive fold than usual; or less frequently by no trace whatever of reduplicated mucous membrane. The many fibres and trabeculæ of mucous membrane which occur out of the position of the more constant folds seem to have no order in their distribution. The constant folds show considerable diversity in the various tympana, on account of the variability of their place of attachment, which admits of a change of position within certain limits. Even the two tympana of the same individual are not alike, in spite of marked common characteristics.

In the fœtus at term, the mucous-membrane reduplications, as a rule, are more succulent and numerous than in the adult; and it is especially noticeable that those mucous folds which were present were perfect, almost without exception, and not represented only partially, as is often the case in the adult. In the specimens from very old people there seems to be some tendency to still further atrophy of the reduplications of mucous membrane.

In determining whether the tympana examined were normal or not, great care was used, and all were considered pathological that showed any pathological change, such as thickening or injection of the mucous membrane, opacity, perforation, or cicatrization of the membrana, or impeded movement of the ossicles. It was found that no appearance of the membrana tympani, except the evidence of suppuration, was any indication of the condition of the mucous folds within the tympanum; except also, perhaps, that tympana having very thin, transparent membrane, often contained a noticeably smaller number of mucous folds.

For a more complete account of the tympanum than is given in this article, I refer the reader to Professor Politzer's "Zergliederung des Menschlichen Gehörorgans," 1889, which is the only complete work published on this subject.

The mastoid antrum was found to contain mucous reduplications in 51 cases out of 108 observed; the reduplications were abundant in 29 of these (Figs. 2, 3, and 4). There was usually a central longitudinal band or nucleus from which radiated fibres and tuberculæ; and sometimes the main axis of these folds was carried forward on to the back of the incus and the head of the malleus, joining the mucous system of these ossicles. Not infrequently there were only a few scattered irregular fibres in the antrum or at its mouth; and sometimes there were in it some scattered trabeculæ of mucous membrane, resembling very much in their arrangement the osseous trabeculæ of the mastoid cells, and seeming to be a continuation of them.

The posterior ligament of the incus is composed of an outer and an inner more or less fan-shaped band of glistening tendinous fibres given off from the sides of the tip of the short

process. The origin of the outer band is the top of the outer surface of the incus, and this part of the ligament in the adult is directed upward about 45° from the horizontal. The inner band of the ligament is often very short; in the adult it is nearly horizontal and arises from the bottom of the inner surface of the incus; this means, in other words, that these two bands of the posterior ligament of the incus are not symmetrically placed about the tip of the short process.

From the outer part of the posterior ligament of the incus two mucous reduplications extend forwards: one or both of them were found in 115 out of 116 observations and the space external to the incus was wholly occluded by these folds in 89. The lower, or inferior external fold is almost horizontal in the adult, and extends forward to the posterior border of the external ligament of the malleus, and from about the median line of the incus to the lower margin of the niche in the external wall of the tympanum above the membrana (see Figs. 1, 5, and 8).

The upper, or superior external fold is much higher along its outer and anterior border than its inner and posterior border: its plane varies in obliquity from 45° to 90° in the adult. Its inner border follows the outer border of the dorsal aspect of the incus; crossing over the crest of that bone, it sometimes joins the suspensory ligament of the malleus, or a mucous fold descending outwards from this ligament; and sometimes, the anterior end of the mucous fold not rising so high, skirts round the anterior surface of the head of the malleus and joins a mucous fold descending anteriorly over the head of the malleus; and sometimes, not going so far forward, it has a free anterior border. This fold, when perfect, forms a roof slanting inwards over the space external to the malleus and incus. Thus any fluid flowing forward from the mastoid antrum would naturally be turned inwards by it and flow down the inner side of the malleus and incus (see Figs. 2, 9, 11, and 13).

The suspensory ligament of the malleus is the most variable of all the ligaments; it may be clearly defined or indistinguishable from the adjacent mucous reduplications; it

may be short or long, round or flat. The ligament takes its origin from the anterior end of a ridge of bone which extends along the tegmen mastoid and part of the tegmen tympani, and was first described by Politzer, who called it the spina tegmen mastoid. The direction of the suspensory ligament in the adult is vertical; but in the fœtus at term it slants downwards and inwards at an angle of about 35°. At this age it is often surrounded by a mass of mucous membrane, which extends back on to the incus and conceals nearly the whole head of the malleus. It is attached to the head of the malleus at a point which in the adult is its highest part. This is not the end of the long axis of the malleus, which passes through the head and manubrium, but behind and to the inside of it, and is very close to the articulation of the incus; it corresponds very nearly to the end of the axis which passes through the head and neck of the malleus. The fibres of the ligament spread down the inner side of the head of the malleus, and backwards oftentimes on to the incus.

The anterior fold of the malleus is one of the most constant mucous reduplications. It starts from the suspensory ligament of the malleus and goes forwards over the head of the malleus and down, joining the top of the anterior ligament. Its superior border is attached to the tegmen tympani, and it there forms a nearly vertical curtain. This fold was found in 112 tympana out of 113, and was perfect in 81 of them (see Figs. 1, 4, 5, 6, 8, and 14).

A less constant fold, the external fold of the malleus, goes outwards and downwards from the suspensory ligament towards the external ligament of the malleus. This fold was found in 55 tympana, and perfect in 46 of them, out of a total of 102 tympana examined (see Figs. 5 and 6). Sometimes an irregular mass of folds is found in front of, and external to, the suspensory ligament (see Fig. 13).

There is a rather strong fold of mucous membrane of frequent recurrence attached to the head of the malleus, having a free internal border. This internal fold of the malleus is nearly horizontal, but slightly higher in front. It stretches between the lower anterior and inner borders of the head of

the malleus back as far as the lower toothed process of the incus, the anterior ligament, and the external end of the spina transversa tympani. Near the spina transversa it often joins the superior tensor tympani fold coming from the tensor tympani tendon (see Figs. 1, 6, and 9). The internal fold of the malleus was found in 37 out of 40 observations.

The capsule of the tensor tympani tendon is often very strong and is usually prolonged forwards into a band of glistening fibres, which becomes broader externally and is attached along the base of the processus gracilis, or, in the absence of this process, to the anterior ligament of the malleus, thus making an internal ligament for the malleus. Occasionally some of the fibres of the tensor tympani tendon, as they approach the malleus, separate from the main bundle and are attached, together with the internal ligament of the malleus, to the processus gracilis or the anterior ligament of the malleus.

From the anterior edge of the capsule of the tensor tympani tendon there are sometimes two folds given off. It is very rare that a part at least of one of them cannot be found. In 111 of the 114 tympana examined, one or both of these folds were found; and in 81 one or both of them were perfect (see Figs. 1, 3, 5, 6, 7, 9, 12, 14, and 15). The lower of these two folds, or the inferior tensor tympani fold, is nearly horizontal in the adult, and extends forward to the roof of the opening of the osseous Eustachian tube. It is attached on the outer side to the anterior ligament of the malleus, and the *cristus* tympanicus (Gruber) anteriorly to the roof of the Eustachian tube; and on the inner side, to the longitudinal projection, symmetrical with the tympanic crest, which is formed by the lower lamella of the canal of the tensor tympani muscle, where the belly of this muscle rounds out. This protuberance seems to be common enough to deserve a name, and might be called *crestus tubæ*. The mucous fold is also attached on its inner side to the processus cochleariformis, and posteriorly to the capsule of the tensor tympani tendon (see Fig. 15). The second, or superior, tensor tympani fold is the more constant. It rises from the front part of the tendon, running forward at an angle of

about 45° or more, and is attached to the anterior ligament of the malleus, the spina transversa tympani, and the processus cochleariformis (see Fig. 15). The spina transversa tympani forms an arch from the spina tympani major to the upper lamella of the processus cochleariformis. I have seen another similar spina a short distance anterior and parallel to this. Anterior to the superior tensor tympani fold, and sometimes bounded below by the inferior tensor tympani fold, there is a variable space, sometimes of considerable size, which might be called the *anterior chamber* of the *tympanum*. In the fœtus at term this space is very small.

The anterior pocket of the membrana tympani is a very small affair, not at all resembling the posterior pocket. It is a truncated pyramid; its apex is formed by a thin, rather delicate membrane, which connects the anterior band of Prussak with the anterior ligament of the malleus, and thus forms the anterior half of the floor of Prussak's chamber. Occasionally this membrane is attached to the membrana tympani, lower than Prussak's band; very rarely this membrane is wanting, and then the anterior pocket of the membrana tympani opens directly into Prussak's chamber. Prussak's chamber, or the space which is bounded externally by the membrana flaccida of Shrapnell, in the majority of cases is wholly cut off from the cavity of the tympanum, or communicates with it only by a narrow opening, which would be readily closed by a slight inflammation. When this space is inflamed and surrounded by a mass of swollen mucous membrane, fluids can escape most readily through the delicate membrana flaccida; and through a perforation thus formed, if the external ligament is imperfect, the region above and external to the head of the malleus also, if inflamed, may find a small outlet for secretions and exudations. The inner wall of the anterior pocket is formed by the spina major and the anterior ligament of the malleus; the posterior wall, by the malleus; the anterior wall, by the annulus tympanicus; and the outer wall, by the membrana tympani. The border of the pocket is formed by the anterior ligament of the malleus (rarely by the corda tympani

nerve, whose course is usually a little higher than the lower border of the ligament), and is either free or runs into the mucous fold of the tensor tympani tendon.

The posterior pocket of the membrana tympani is very variable in size, according to the position of its free border. This varies in level from the base of the manubrium in very small pockets to half-way down the manubrium in very large ones. The fold of the pocket is a very constant mucous fold, enclosing the corda tympani nerve and a strong band of connective tissue (see Fig. 14). This fold of the pocket is nearly parallel to the general direction of the membrana, approaching it above. The ligamentous parts of the fold—for it serves as a posterior ligament to the malleus—is very constant in its occurrence. It resists chiefly forward motion of the manubrium, but also, to some degree, inward motion. I was unable to find any histological resemblance, in the arrangement of the connective-tissue fibres enclosed by the mucous fold, to the connective tissue of the membrana tympani, and see no reason for speaking of the fold as a part of the membrana. The ligamentous part of the fold is composed of strong longitudinal connective-tissue fibres. It forms the free border of the pocket for about its anterior third, and has its sides nearly parallel to the membrana, stretching upwards and backwards. It is attached anteriorly along the inner edge of the posterior aspect of the manubrium, about the level of the attachment of the tensor tympani tendon, or a little below it; and, posteriorly, close to the inner side of the annulus tympanicus, at a spot about on a level with the anterior spine of the tympanum, I have not infrequently noticed, for the attachment of this posterior ligament of the malleus, a small flat spine pointing forwards and projecting in front of the neighboring attachment of the membrana. This spine seems to occur often enough to be worthy of a name, and might be called *spina tympani posterior*, a name more appropriate to it than to the spina tympani major. The posterior two thirds of the free border of the posterior pocket of the membrana tympani is formed by the corda tympani nerve. These two elements of the posterior fold of the membrana

—the ligamentous band and the nerve—cross each other, forming a broadly obtuse angle in the fold of the pocket. The roof of the posterior pocket, like the roof of the anterior pocket, is formed by a thin membrane whose extensor border usually coincides with the corresponding band of Prussak, thus separating the pocket from Prussak's space. In the case of the posterior pocket, this membrane (which is less constant than the roof of the anterior pocket) forms the posterior half of the floor of Prussak's space. It is perfect in the majority of cases, but, when wanting, it allows free communication between the posterior pocket and Prussak's space. It is the exception, not the rule, when the fold of the posterior pocket comes in contact with the membrana. When this contact occurs it is usually at the upper and posterior periphery of the fold, where the fold normally approaches very near the membrana. Instead of a distinct roof to the pocket, the fold may gradually approach and unite with the membrana tympani. When this is the case, the fold is often fenestrated—that is, its principal elements, the posterior ligament of the malleus and the corda tympani, are not united,—and then the posterior ligament of the malleus is generally attached to the manubrium much more externally than usual, and close to the membrana. In the larger number of cases, the long process of the incus is connected with the fold of the posterior pocket about where the corda tympani and the posterior ligament of the malleus cross each other. The connection is either direct or completed by mucous-membrane bands. The boundaries of the posterior pocket are: *superior*, the floor of Prussak's space; *anterior*, the posterior surface of the manubrium; *external*, the membrana tympani; *posterior*, the annulus tympanicus just internal to the sulcus; *internal*, the fold of the pocket. Sometimes the fold of the posterior pocket is carried up above the floor of Prussak's space, and joins the posterior part of the external ligament of the malleus; but this is rare, there being usually an opening into Prussak's space just above the fold of the pocket, and below the external ligament of the malleus.

The external ligament of the malleus is a nearly constant,

horizontal, fan-shaped ligament. The posterior border of the fan is usually longer than the anterior, and is attached to the malleus between the head and neck from the anterior ligament or the outer border of the spina major round to the articulation of the incus. In a few cases this attachment of the ligament is carried backwards across the malleo-incudal articulation, and a few fibres arise from the incus itself. The outer border of the ligament is attached along the border of the notch of Rivini, which causes it to be slightly convex above. This ligament is very variable in strength, but never very strong. The strongest fibres lie sometimes in one part and sometimes in another; it is occasionally fenestrated, and especially near the anterior ligament of the malleus. The external ligament forms the roof of Prussak's chamber, and below its posterior border there is usually an opening into this chamber; sometimes this opening is closed by mucous reduplications from its borders to the outer surface of the incus. The posterior border of the ligament is sometimes continued backwards along the outer side of the incus by a mucous reduplication, or downwards to the upper border of the posterior fold of the membrana. Mucous folds are occasionally attached also to the upper anterior surface of this ligament.

The anterior ligament of the malleus is the strongest of the ligaments; its internal and lower fibres are many times longer than the outer upper ones; it is a flat ligament, having its long diameter nearly vertical, and four or five times as long as the horizontal diameter. It is attached to the malleus, on its anterior side, from just above the attachment of the tensor tympani tendon to about half-way up the head, where its place of attachment is marked by a well-defined depression. The shorter fibres of the ligament end on the inner and lower surface of the spina major, while the longer ones disappear from the tympanum in the Glaserian fissure.

The angle formed by the manubrium and the long process of the incus often has a mucous-membrane webbing, which sometimes extends as far down as the middle of the long process and the upper third of the manubrium (see Fig. 15).

Of the more uncommon folds of the malleus and incus, the following may be mentioned. Perhaps the most frequent of these is the posterior internal fold, a reduplication which is bounded by the posterior border of the long process of the incus, the lower border of the short process, the inner wall of the tympanum, and the stapes, where it is connected with the mucous system of the stapes. Occasionally it just misses the short process, and is attached to the external wall of the tympanum. This fold is almost horizontal, but highest along its external and posterior borders. It was found in 52, and perfect in 20, out of 117 tympana examined.

A fold is sometimes seen attached to the posterior margin of the tensor tympani tendon capsule, the malleus, the lower toothed process of the incus, the anterior border of the long process of the incus, the stapes, and the inner wall of the tympanum. This anterior internal fold is more oblique than the previous one; its external and anterior borders being highest. It was found in 26, and perfect in 11, out of 119 tympani observed (see Figs. 10, 11, and 13). If the previous fold is also present it may join this fold, and form a half-saucer-shaped curtain across the tympanum. Its lowest point is just above the stapes. These two folds together, especially when the posterior one is attached externally to the external wall, just above the membrana, form a curtain, covering in all the ossicles except the manubrium, and making the tympanum, in this respect, remarkably like the tympanum of the dog. The round window may also be free. I found the ossicles shut off from the cavity of the tympanum in this way in 9 tympana out of 115 examined (see Fig. 10). These two internal folds, in conjunction with those external to the malleus and incus, and those anterior to the tensor tympani tendon, divided the upper from the lower part of the tympanum wholly in 9 tympana out of 115 examined, and more than half in 40 (see Figs. 10 and 11). Sometimes a fold rises from the stapes, and joins the inner surface of the long process of the incus to the inner wall of the tympanum, forming the vertical fold of the incus, which occasionally rises to the tegmen, along the inner posterior side of the head of the malleus. It was present in 20 and perfect in 3 out of 39 tympana examined (see Fig. 13).

A rare fold rises from the tensor tympani tendon sheath, along the inner surface of the malleus, to the tegmen and the inner wall of the tympanum. This fold was found in 6 and perfect in 2 out of 41 tympana examined.

I have seen the stapes only three times without some mucous reduplication. In 109 observations they were present in 106 tympana.

Of the upper and lower sides of the stapes, that which is furnished with the more abundant mucous membrane is usually the more adherent to the adjacent wall. Whether this is due to the presence of so much mucous-membrane adhesions, or to the propinquity of the stapes to the wall, is doubtful; but the latter seems to be the cause, for, in the majority of cases, the stapes seems to be more adherent to the lower wall of the pelvis, and we find that the foot plate of the stapes is usually in closer relation with this wall than with the upper wall, for the lower wall rises more abruptly from the edge of the fenestrum ovale. Out of 109 observations, the stapes was more adherent below in 78, and more adherent above in 7. The mucous membrane, connecting the stapes with the walls of the pelvis ovalis, was either a few fibres or bands, or a web of mucous membrane connecting the whole length of one or both crura to the wall. The upper side of the stapes sometimes had fibres and folds connecting it with the upper wall of the pelvis, but the closest connection was found below.

The membrana propria of the stapes showed great variations. I found only one specimen in which this membrane was double, each half being flush with the corresponding edge of the crura, and completely enclosing a space. Sometimes there was one membrane stretched between the upper or lower edges of the crura,—usually the lower—and a partial webbing of the opposite edge of the crura. Very rarely there was no trace at all of a membrana propria. Occasionally the membrana propria seemed to have grown on to the lower wall of the pelvis ovalis. In the majority of cases a mucous fold stretches across the space which the tendon of the stapedius muscle bridges. There is also a fold similarly arranged in connection with the bony spiculæ, which occur

below the stapedius tendon, and are nearly parallel to it. In the fœtus at term there are usually two of these spiculæ, but in the adult the lower one has usually disappeared. Sometimes the lower one is left and the upper one gone, or both are wanting.

The small depression, or sinus tympanicus, in the inner wall of the tympanum, just below and behind the pelvis ovalis, is sometimes covered by a mucous-membrane fold. The pelvis rotundum is not infrequently obstructed by mucous reduplications, and is occasionally closed by them. These are either fibres or folds. When folds, they are apt to be arranged in one of two ways,—either like a star with its rays attached to the walls of the pelvis rotunda, or in a ring of mucous membrane about the opening of the round pelvis—having a free opening through its centre to the fenestra rotunda. Out of 99 observations, 44 had the round pelvis at least partially occluded (23 completely so) and 55 were perfectly free. The inner and posterior walls in the vicinity of the fenestra are not very infrequently provided with small webs of mucous membrane connecting some of the irregularities of the surface, but I found these folds in those tympana only having an abundance of mucous reduplication in outer parts.

Besides these folds and fibres of the tympanum, which appeared to me to be susceptible of classification, I not infrequently met some that could only be described individually (see Figs. 14 and 15).

In a few of the tympana I noticed a tendency of the bone of the wall of the tympanum to extend a short distance into the mucous fibres and folds.

It is sometimes stated that the folds of the membrana tympani, which are slight convexities of the surface, going forward and backward from the short process of the malleus below Prussak's fibres, are due to the outward pressure of this process. But it seems to me that the folds are due to the change in the tension of the circular fibres of the membrana, as it passes from the membrane proper to the membrane of Shrapnell.

FIGURES.—All the figures illustrating this article were

drawn from nature and are several times the natural size. The mucous-membrane reduplications are indicated by color. The lightest colored one is the nearest to the eye.

FIG. 1. Fœtal tympanum at term opened from above; showing the inferior external fold. The lower part of the anterior fold of the malleus can be seen on the upper surface of the malleus. The internal fold of the malleus lies in front of the head of the malleus, and nearly crosses the tensor tympani fold, which can be just seen on its inner side.

FIG. 2. Adult tympanum, viewed from above and behind. Mucous folds extend forwards from the mastoid antrum on to the ossicula. There is a superior external fold, and abundant folds about the head of the malleus. This is the same tympanum as in Fig. 12.

FIG. 3. Adult tympanum, the same as in Fig. 15, looking forward, downward, and outward. A mass of folds extends forwards from the mastoid antrum on to the ossicula. The anterior fold of the malleus can be seen, also the tensor tympani fold. A fibre joins the head of the malleus to the internal wall.

FIG. 4. Adult tympanum, the same as in Fig. 9, looking outward. The anterior fold of the malleus is seen, also folds extending from the mastoid antrum. A part of the superior external fold can be seen in the background, and in the foreground a part of the tensor tympani fold.

FIG. 5. Adult tympanum, viewed from above. Anterior to the head of the malleus, and reaching up over it a little, the external fold of the malleus can be seen going outwards to join the inferior external fold. The lower part of the anterior fold of the malleus is seen. The tensor tympani fold is seen in an imperfect condition.

FIG. 6. Adult tympanum, opened from above. Anterior to the inner surface of the head of the malleus, the internal fold of the malleus can be seen. The lower part of the anterior fold of the malleus is external to this, and external to this again is a remnant of the external fold of the malleus. The external folds are unusually imperfect, and reduced to a small web attached to the posterior ligament of the incus. The tensor tympani fold is in the anterior end of the tympanum. A fibre extends from the body of the incus to the inner wall of the tympanum, which is a part of an imperfect posterior internal fold.

FIG. 7. Adult tympanum, opened from above. On the inner side of the head of the malleus a mucous web extends along the

posterior edge of the tensor tympani tendon, and the anterior surface of the long process of the incus. This is really an imperfect anterior internal fold. Anteriorly lies the tensor tympani fold. The external fold appears of irregular level.

FIG. 8. Adult tympanum, the same as in Fig. 14, viewed from above. This shows the inferior external fold, the tensor tympani fold, and the anterior fold of the malleus rising obliquely outwards.

FIG. 9. Adult tympanum, the same as in Fig. 14, viewed from above. This shows the posterior internal fold; also the posterior part of the superior external fold, the anterior part having been removed. The anterior and the internal folds of the malleus can be seen, and a very small part of the tensor tympani fold.

FIG. 10. Adult tympanum, from the same individual as Fig. 11, viewed from below. This shows a completely divided tympanum; the ossicles, except the manubrium, are cut off by a curtain of mucous membrane. The anterior and the posterior internal folds are shown.

FIG. 11. Adult tympanum, from the same individual as Fig. 10, viewed from above. This shows the anterior and the posterior internal folds; also the superior external fold, the anterior fold of the malleus, and an unusual internal fold of the malleus. Here there is complete horizontal division of the tympanum.

FIG. 12. Adult tympanum, the same as in Fig. 8, viewed from below. This shows the tensor tympani fold, the internal folds, the webbing of the malleus and incus, and the vertical fold of the incus.

FIG. 13. Adult tympanum, viewed from above. This shows the top edge of the vertical fold of the incus; also an irregular mass of mucous membrane in front of the head of the malleus, the anterior internal fold, and the external fold.

FIG. 14. Adult tympanum, the same as in Fig. 8, looking outwards. This shows the fold of the posterior pocket of the membrana tympani; also mucous fibres occurring in irregular positions, the anterior fold of the malleus, the tensor tympani fold, a part of the external fold, and the webbing between the malleus and the incus.

FIG. 15. Adult tympanum, the same as in Fig. 3, viewed from below. This shows the inferior tensor tympani fold, which is here perforated, and through the perforation the superior tensor tympani fold. Irregular mucous-membrane fibres are shown, and mucous-membrane webbing between the malleus and the incus; also the posterior internal fold.

CHOLESTEATOMA, PERFORATION OF SHRAPNELL'S MEMBRANE, AND OCCLUSION OF THE TUBES—AN ÆTIOLOGICAL STUDY.

By Dr. FRIEDRICH BEZOLD of Munich.

Translated by Dr. J. A. Spalding, Portland, Me.

IT is now eleven years since I published a paper on the desquamative processes of the middle ear,[1] and promised another article on *the manner in which concentric epidermic masses at this spot originate;* but, strange to say, the difference of opinion between pathological anatomists and practical otologists on this point exists just the same to-day as it did then.

The contrast between the results of pathological anatomy and clinical observation has very lately obtained a renewed and more decided emphasis, owing to a discussion that followed a paper of Prof. Kuester at a recent meeting of the Berlin Medical Society, where Virchow authoritatively asserted his belief that cholesteatoma of the temporal bone is a heterologous tumor, just as he had done years before in his fundamental book on pearly tumors (cholesteatomata), in which he reviews his entire pathological and anatomical experience.

I will here briefly call attention to Virchow's experience in this province, extending over 24 years, embracing 15 cases of cholesteatoma of the temporal bone and 38 fatal cases of caries of the middle ear without cholesteatoma, showing that the latter amount to about 28 per cent. of

[1] *Arch. f. Ohrenhlkde.*, xiii.

all the fatal cases. Even this number may be too small, because, as Virchow suggests, some of the cholesteatomata may have been previously destroyed by the suppuration. The large percentage of fatal cases has evidently impressed Virchow with the extreme clinical importance of these tumors, for at the discussion in question he advises Kuester, and in another place [1] urges Kuhn, to consider the question of a total extirpation whenever the diagnosis can be made properly, and sufficiently early.

When we come to the pathogeny of cholesteatoma of the temporal bone, we shall be obliged, according to Virchow, to decide in every individual case, whether we shall treat it as a special sort of tumor, or whether we shall align it with the nearest related tumors—on the one hand with atheroma, or on the other with dermoid.

The development and form of cholesteatomata prove sufficiently that they cannot originate from the hair-sacs or from the glands of the external meatus as Toynbee once asserted. "They are, on the whole, rather located in the bone, and particularly in the newly developed cavity," is one of Virchow's recent remarks. He also argues against their relationship with dermoids, "because the enveloping membrane of pearly tumors is the very finest structure that we can discover anywhere." Furthermore, he has not personally witnessed the formation of dermoid in the pia mater, "but on the contrary, half a dozen cases of pearly tumor in basilar sections of the pia." Consequently, he thinks it very curious, "that cholesteatomata in the brain should affect a region lying so very close to the petrous portion," the region about the medulla oblongata and the pons, "so that all this would seem to show that there is probably some connection between these tumors and the pearly tumors of the middle ear."

Virchow thinks that the idea that cholesteatomata originate from an epidermoidal transformation of the mucous membrane of the middle ear is annihilated by the very localization of these tumors, which up to this time he has never been able to discover actually within the tympanic

[1] *Arch. f. Ohrenhlkde.*, xxvi.

cavity itself. On the contrary, he usually discovered the growth "at the upper and outer corner of the tympanum in the direction of the so-called cells of the mastoid process"; "the region at which, in place of air-containing cavities, compact bone develops at a later period of life." "Nevertheless there is a trace of probability that the development of epidermoid masses might have a certain connection with these processes."

Virchow mentions another possibility though he does not lay much stress upon the same; he points at the persistent fissure in the upper portion of the annulus tympanicus. "We might imagine that during the development of the annulus tympanicus, or during its transformation into a portion of the external meatus, a deviation upward and outward occurred at this spot, and that, as it occurred, disturbances arose in its enveloping epidermoidal layer, whereby portions of the membrane split off and became especial nuclei for neoplastic tissue." Then he adds: "I will not go so far in these preliminary remarks as to assert precisely how we may be able to explain satisfactorily the original point from which cholesteatoma arises." In conclusion he remarks that "these tumors have been observed additionally in the frontal bone, the inferior maxillary, the occipital bone, but never, to my knowledge, in any bones of the trunk or extremities."

Now all of these theories advanced by Virchow stand out in bold relief against the practical experiences of otologists in this branch of pathology. We, as specialists in diseases of the ear, can observe these structures not only on the cadaver, but we have been able to follow them out in the living from the very beginning, ever since v. Troeltsch furnished us with the means of properly inspecting the depths of the organ of hearing. Personal experience for many years in a comparatively narrow branch of medicine, to which Virchow is somewhat of a stranger, leads me now to oppose some of his later-day opinions.

v. Troeltsch was the first to deny that cholesteatoma is a tumor, and to assert, on the contrary, that it is a sort of inflammatory product heaped up in the middle ear. Fur-

thermore, he was the earliest observer to emphasize to otologists its threatening aspect when associated with suppurative and septic processes in the middle ear. Since then a large number of cases of this nature have been observed, and numerous cases are on file to facilitate the comprehension of the existence within the middle ear of masses closely resembling epidermis.

The frequency of cholesteatoma in the temporal bone is much greater, according to the experience of otologists, than would seem from the reports of the anatomists; because only a small percentage of cases terminate fatally, whilst the practising otologist discovers cholesteatoma the oftener and the more carefully he searches for this tissue during the course of suppurative inflammation in the middle ear.

My note-books from 1881 to 1886[1] show 34 cases of cholesteatoma in 1,337 cases of chronic otitis purulenta, but this small number is not enough to indicate the precise occurrence of epidermis in the middle ear, because I have only classified under cholesteatoma those cases in which I was actually able to pry out into the external meatus from the middle ear masses from the size of a bean to that of a cherry that had not before been visible in the meatus. There was also a large number of cases of smaller clusters or lamellæ of epidermis of various sizes which were removed from openings in the *Mt* or from apertures in the wall of the external meatus which led into the middle ear. This was additionally so in a large majority of the 88 patients affected with *chronic suppuration of the middle ear accompanied with perforation of Shrapnell's membrane*, and further still in a large number of chronic aural affections, especially those with fistulæ opening into the external meatus or upon the outside of the mastoid process. In every one of these cases epidermis was undoubtedly located in the middle ear, and it is very probable, in addition, that these numbers would be indefinitely increased, if it had been possible to examine the ears pathologically in the above series of patients.

This incredible frequency of a so-called heterologous neo-

[1] *Arch. f. Ohrenhlkde.*, xxi. and xxv.

plasm, and that too at a locality where the occurrence of all the other well-known and undeniable heterologous tumors is one of the greatest of rarities, must naturally arouse considerable doubt and suspicion when the cholesteatoma is classified under the heterologous group.

The reasons which have apparently forced Virchow to this opinion, are the *locality* of the neoplasm, and its *complete heterology* in relation to the locality in which it has developed.

Now so far as the locality is concerned, Virchow says, in opposition to Toynbee's assertion that these tumors originate from the glands and hair-sacs of the external meatus and represent nothing but an atheroma of these structures: "They are actually situated within the bone, in a neoplastic cavity, so that when the material within is macerated, a great cavity is seen as if excavated from the temporal bone."

This is quite true, and agrees with the picture of the bone as seen after the longer-standing cases of cholesteatoma.

Then again, what Virchow says in regard to the place where they develop agrees with the observations of most aural surgeons: "the upper and outer angle of the tympanum toward the so-called cells of the mastoid process," and later: "directly above the upper fissure of the annulus tympanicus, from the upper end of which, however, they generally arise only by constriction." "Generally," he adds, "the cavity filled with the pearly mass lies somewhat outward and upward, so that when we saw perpendicularly from above downward, we obtained the same strata: above, the pearly tissue, next caries, and below, the parts at least relatively normal."

This sketch is very correct, but we cannot grant that this cavity, bisected in perpendicular sections even when falling through the temporal bone at some distance laterally from the pole of the *Mt*, is neoplastic. For, in this region, developed to about the same size in the new-born as in adults, there lies above the innermost portion of the meatus and the tympanum, the Aditus ad antrum, which, still to some extent remaining above the meatus, extends directly into the antrum.

The only way in which we can gain any clear idea of the mutual relation of the meatus, the tympanum, the continuation of the latter upward and backward into the Aditus, and of the Aditus itself, is from casts of these cavities made on fresh temporal bones. In this way only can we represent all these parts simultaneously, and in intimate relations with one another, and thus study them together, their separate regions, and their whole, as one. Plate III. of my "Corrosions Anatomie des Ohres," Munich, 1882, exhibits four such casts, and proves amongst other things that the cavities of the Aditus and the antrum remain throughout close to the upper wall of the meatus, and farther backward close to the upper and posterior walls of the same, and never more than ½ *cm* distant. Laterally, this propinquity extends almost to the external termination of the osseous meatus. A temporal section made perpendicularly through the latter will therefore, when close to the *Mt*, lay bare the Aditus, but if placed farther backward, it will expose the antrum at the same time with the lumen of the meatus.

The experience of practical otologists shows that these two cavities in both the living and the dead are the most frequent seat of cholesteatoma. If the accumulations of material have worn away the parts by pressure and attrition, we find the Aditus and antrum eccentrically enlarged, pressed still closer toward the meatus, and when the masses are inordinately large, even communicating over a large surface with its lumen, the intervening bony wall having entirely disappeared.

Aurists have expressed various opinions concerning the *origin of epidermis at this locality.* That of v. Troeltsch is the one most widely known; having observed flat giant-cells resembling epidermis in the lining membrane of the antrum, he thinks it probable, that cholesteatoma is a collection of inflammatory superficial products, or, in other words, "the sequence of a former or still persisting suppuration from the middle ear." This author also lays special stress upon the "large pus nuclei" so often seen within cholesteatoma; "the pressure which they exercise upon the walls of the cavity produces not only an abundance of cellular

products, but these are altered in form in such a way that they greatly resemble stratified epidermis."

Again, in a somewhat different line of thought, we observe the uniform extension of a metamorphosis in the epidermis of the entire cavity, and the persistence of the same even after all inflammatory irritation has terminated. The epidermis in cholesteatoma of the temporal bone does not differ at all, at least in the external layers, from the horny layer of the cutis, provided that it is protected from desiccation and kept uniformly moistened, as happens within the spaces of the middle ear. Even the macroscopic description of cholesteatoma from both the living and the dead makes it plain that it must have originated from a large surface, frequently from the entire extent of the walls of the bony cavities, the smooth surface perfectly reduplicating the form of the cavity, which is often indented. Even with the naked eye, and especially toward the periphery, we observe perfectly concentric layers, which are so thin and so regularly arranged that, according to Johann Mueller, it is this very parallel stratification that causes the interference of light and the mother-of-pearl reflex upon its surface. Toward the antrum, the strata more frequently interlace with one another, or lie over and between one another, and are of a dirty-brownish hue under the microscope, separating from one another the flat non-nucleated epidermis cells, with a large mass of glittering irregular clumps, also covered abundantly with crystals of cholesterin. Then again, over a greater or less space we find in a large majority of cases genuine cheesy and excessively fetid masses, in which we no longer see epidermis cells, nor even pus cells, but simply large masses of fat-needles, detritus, the clumps before mentioned, and monstrous masses of bacteria.

Here we have the oldest central portions of the accumulations, already fallen into disintegration. Their central position, however, by no means perfectly protects them from external influence, as we often enough have an opportunity to see in the living. For whenever we succeed in reaching the cholesteatoma with a syringe from without, through an opening either in the *Mt*, or in the walls of the meatus, we

first see these characteristic cheesy masses extruded from the middle ear by the force of the current, whilst only gradually, and often only after energetic injections, continued sometimes for many weeks, do we see fresh and white cholesteatoma escape. Direct injections into the orifice with Hartmann's bent tympanic tube are generally indispensably necessary to complete their entire removal. When the orifice leading into the meatus is large enough, we can, finally, though often only amidst severe inflammatory symptoms, succeed in forcing out the entire cholesteatomatous mass through the opening in the wall of the meatus and evacuate portions as large as a hazel-nut, or even a walnut.

The remaining cavity can often be fully seen through the external meatus, and still more perfectly through the newly formed tissue when the rupture has taken place through the external wall of the mastoid, and after the inflammatory and suppurative symptoms have terminated, the cavity is lined within with a white or pinkish membrane perfectly resembling a thin layer of cutis, and without a trace of fluid secretion, providing that the suppuration does not still continue at a more distant portion of the middle ear.

We have an instructive case of this sort reported by Steinbruegge [1], in which, after evacuation of its cholesteatomatous contents, an extensive cavity could be overlooked through a large fissure in the outer wall of the mastoid bone.

Additionally, I have described,[2] after removal of a large cholesteatoma, a nearly perfect view of the cavity through the defective posterior superior wall of the meatus. This cavity rapidly diminished in size from secondary inflammation of the entire mastoid bone after the removal of its very extensive contents, and its walls are now covered with a thin, whitish, perfectly dry lining membrane. The wide communication with the meatus also allowed me by a method described in the paper to measure the capacity of the cavity, which amounted to 2.85 *ccm*, including the meatus, and 1.95 *ccm* for the cavity alone. Thirteen years have passed since the opening was healed, and there has been no

[1] These ARCHIVES, ix., 21, and similar cases, these ARCHIVES, x., 227.
[2] *Arch. f. Ohrenhlkde.*, xiii., 31.

change in the appearance of its walls. There has never been any re-accumulation of epidermis, and the lining membrane is precisely like that of the cutis of the meatus and *Mt*, into which it extends without interruption.

Hummell has also collected [1] several other cases of this nature from my note-books, and Barth mentioned at the discussion which we are now noticing, a case precisely like that of Steinbruegge's.

When we examine on the cadaver the lining membrane of those cholesteatomatous cavities that have not proliferated, we generally find a minimal layer of connective tissue and a delicate rete Malpighii of several strata, the uppermost of which pass directly over into the non-nucleated flat epidermic layers which compose the cholesteatoma itself.

Proliferations are on the whole met with quite as often in the living as in the dead, for the reason that those who suffer from the disease only seek advice and assistance when the accumulation hitherto latent has produced excessive symptoms of irritation and inflammation, and, additionally, because those persons seen on the post-mortem table, or at least those that come under the observation of the otologist, have lost their lives from suppurative processes excited by the cholesteatoma itself and its sequences.

There is no doubt that the same metamorphosis and formation of a rete Malpighii occurs frequently enough on the epithelial covering of these proliferations, as Lucae has actually demonstrated,[2] but these proliferated patches are undoubtedly the exclusive locality for the origin of those strata of epidermis which stretch uniformly over large tracts, or over the entire cavity.

The antrum and Aditus occasionally exhibit a genuine thin cutis with all the peculiarities of the latter. The locality of the cubical mucous-membrane epithelium in one layer is occupied by a rete Malpighii of several layers, which exhibits all the peculiarities and characteristics of the real cells of a rete elsewhere. When the cavity stands wide open outward, its surface is not moist like the mucous membrane,

[1] *Arch. f. Ohrenhlkde.*, xxiv., 203.
[2] *Arch. f. Ohrenhlkde.*, vii., 255.

but dry, and much resembling the skin of the human body. If closed more or less completely, its surface is more abundantly exfoliated, but the products which it furnishes vary totally from those of normal or inflamed mucous membrane, being almost identical with those of the cutis whenever a portion of this, as is the case in atheroma or beneath the prepuce, etc., is shed off and exposed to permanent moisture.

Wendt [1] explains the formation of a rete Malpighii upon the hypertrophic mucous membrane of the tympanum as the result of a desquamative inflammation of the mucous membrane.

Politzer ("Lehrbuch," 2te Aufl., p. 316) thinks that a part of the cholesteatoma is due to "the snaring off of gland-like epidermic-covered cavities in the proliferated tympanic mucous membrane," in which he has "repeatedly discovered enclosed, small, white, and smooth epithelial globules as large as a pin-head."

All of these theories, however, do not bring us a bit nearer the actual solution of the riddle—why genuine epidermic strata can develop upon the outer surface of remote muco-membranous regions; and this shows us why the idea of an *embryonal preformation of cholesteatoma*, as suggested by v. Buhl and lately supported by Kuhn and Kuester, has found ardent defenders.

The simplest explanation of the heterotopia of cholesteatoma appears to me to lie in an occurrence which we witness daily in an occlusion of middle-ear suppuration.

When suppurative inflammations in the tympanum with loss of substance in the *Mt* have come to an end, the lining membrane of the region affected offers various appearances depending upon the extent, and still more upon the situation, of the perforation that has remained. Generally, however, there are two especial conditions of the tympanic mucous membrane, which, as we inspect them upon our patients, reveal here and there extraordinary variations and the greatest percentage of frequency. Thus the naked mucous membrane of the inner wall of the tympanum looks perfectly or nearly normal, somewhat of a bony-yellow,

[1] *Arch. f. Heilk.*, xiv., 428.

or with a mixture of whitish-gray, slightly moistened, with a shining surface, and with many irregular and mostly shining reflexes, here and there overrun with separate vascular ramifications.

Now this state of things is found when the perforation is central, even when it has extended far toward the periphery, and even when the entire manubrium has been eaten around, provided that it still lies free in the open space of the perforation, and that the extreme peripheral margins of the perforation do not anywhere touch the inner tympanic wall.

The condition of affairs is, however, entirely different when any bridge has formed between the remnants of the *Mt* and the mucous membrane of the tympanic wall, or when, additionally, the limbus of the *Mt* has succumbed to the destructive action of the previous suppuration.

The moment that the sharp distinction between the cutis and the mucous membrane has once been lost by the destruction of the surface at any spot, we see the cutis of the remaining intact region gain the ascendancy over the mucous membrane, and extend with much greater rapidity over the entire district.

So long as there is the least trace of a *Mt* toward the extreme periphery, it seems to offer a safe protecting bulwark against the advances of the cutis over the surface of the tympanum. But when this margin is once gone, or bridged over, then the epidermic layer can extend over a large expanse of territory, according to Schwartze, as far as the cells of the mastoid process ("Lehrbuch," p. 184). This preponderance of the epidermis is easy to understand from the conditions of its growth, which we are able to study, particularly in the *Mt* and upon the walls of the meatus.

Thus blood extravasations, crusts, etc., which cling firmly to the surface of the *Mt*, visibly retreat beneath our watching eyes, as we well know, gradually passing toward the periphery, where they can often be followed in their wanderings outward as far as the extreme end of the osseous part of the external meatus.

I also have a set of epidermic ribands, which, rolled up in wads and impregnated with cerumen, totally filled the otherwise intact meatus, and were removed by the use of the

syringe. A portion of one band, from ½ to 1 *cm* wide, measured 7 *cm* in length, more than double that of the entire external meatus.

We thus prove that the epidermis, at least that of the *Mt* and meatus,[1] exhibits a surface growth that has never yet been observed in a similar manner, so far at least as my knowledge goes, in the epithelium of the mucous membrane.

This bridging over is most distinct between the *Mt* and the tympanic wall, where the manubrium lies high aloft and isolated in the perforation, or where at the same time it has rested upon the same wall, with a projecting portion of the marginal perforation, and then attached itself. The posterior half of the upper margin of the perforation is the one that agglutinates itself with the greatest ease to the long process of the incus, to the stapes and its tendon, toward which, or even in actual contact with which when the lower half of the *Mt* has been destroyed, this margin has been dragged by the preponderance of the tensor tympani. Nevertheless, we may additionally observe equally extensive adhesions between the lower, anterior, and even posterior margins of the perforation and the adjacent tympanic wall; all of which are, however, extremely difficult to see in their entirety in the living, owing to the extreme perspective foreshortening that their position causes them to assume. The post-mortem examination, moreover, shows us that the entire margin of the perforation, exclusive of the manubrium, may often form a total circular adhesion with the neighboring tympanic wall. And further still, by inflating the Eustachian tube on the cadaver, I once found the tympanic orifice of the tube almost entirely occluded by adhesions with the adjacent margin of the perforation, a fact demonstrated by the rest of the *Mt* puffing up all around the tubal orifice as the inflation was repeatedly performed.

The mucous membrane in the region of these bridges between the *Mt* and tympanum has also entirely altered in

[1] I do not know whether similar growth has been shown in other portions of the epidermis. The persistence of tattooing stains in the locality where they have been placed, seems to negative such a growth; nevertheless, so far as we know, the coloring matter might originally have been forced beneath the migratory layer of the skin.

character over such extents of space as we are able to view in the living. The difference in appearance between the external surface of the *Mt.* and the tympanic wall has totally disappeared, both forming a continuous whitish-gray, dull, dry surface, and it is only from the irregular formation of the surface, and from cavities which lead deeply down, partly beneath the manubrium and partly beneath the peripheral bony wall, as well as from the immobility when using Siegle's pneumatic speculum, and the bony hardness perceived when we use the probe, that we are able to convince ourselves, despite the appearances, that it is actually the tympanic wall that lies before us.

It is not always possible to draw a sharp line between the remains of the suppurative process and extreme atrophy of the *Mt* with extensive synechiæ. Both processes are really much alike, since in the latter the tubal affection, which is the fundamental cause of the whole, often gives rise to repeated though generally brief periods of suppuration, with rupture of the atrophic spots and consecutive adhesions inward.

We are especially able to follow out in all its consequences the gradual extension of the formation of epidermis when greater defects at the inner end of the bony meatus permit us to obtain an extensive view of the spaces of the middle ear. Wherever this process is still on its onward march we often find, between the whitish mucous surfaces already coated with epidermis and the slightly red proliferating mucous surfaces, a sharply defined zone in the shape of a white margin of epidermis. Thus, in a case of Hummell's, where a large portion of the inner surface of the entire middle ear lay bare for inspection, owing to an extensive defect in the upper wall of the external meatus, we could clearly see the whole process going on before our eyes, the advancing expanse of epidermis, and the cutting off of islands of mucous membrane until the whole surface had become almost entirely epidermatized. And in another case that I followed up to the end of the affection, in which almost all of the bony labyrinth had been exfoliated,[1] the cavity resulting was almost entirely lined with epidermis;

[1] *Zeitsch. f. Ohrenhlkde.*, xvi.

whilst finally in another case six years after the exfoliation of a sequestrum representing the greater portion of the pyramid, the enormous cavity embracing the meatus and tympanum and reaching to the outer wall of the mastoid yielded to the force of the syringe large stratified scales of epidermis, which had all the characteristics of a genuine cholesteatoma.

Here then we have after destructive inflammations examples of bony cavities in the temporal bone, upon the walls of which epidermis had abundantly accumulated in characteristic layers.

The great majority of cholesteatomata cannot, however, develop in the fashion thus depicted, because in the case of these tumors perforations in the *Mt* are small, or even entirely absent. Nevertheless there is a preformed cavity, appertaining to the middle ear though somewhat distant, and this is the Aditus and the antrum itself, in which Virchow and v. Troeltsch and all following observers have most frequently seen cholesteatomata.

Still, as Habermann's lately published post-mortem examinations show,[1] a band-like extension of a strip of cutis may grow even from the lower end of the manubrium into the antrum, and there become the matrix for a genuine cholesteatomatous formation. Habermann has for the first time offered the *microscopic proof* that in his case a genuinely developed rete Malpighi could be followed in its continuity from the starting-place into the antrum.

The origin of the majority of cholesteatomata has gradually become clearer to me since I have paid greater attention to a former neglected disease, the perforative inflammation of Shrapnell's membrane, and have discovered in numerous post-mortem examinations both a perforation at this region and the presence of cholesteatomata.

Moos remarked the rarity of perforations in Shrapnell's membrane as far back as 1866,[2] and Schwartze, in 1878, depicted[3] a cicatrized perforation at this spot, without attaching to it any great pathological interest.

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[1] *Arch. f. Ohrenhlkde.*, xxvii., p. 42.
[2] "Klinik der Ohrenkrankheiten," 126.
[3] "Path. Anat. d. Ohres," p. 60.

It was only after I had published the account of a fatal case of pyæmia and meningitis from middle-ear suppuration accompanied with perforation of Shrapnell's membrane that the idea of the serious danger of these suppurations to life first occurred to me. I observed how obstinate many of them were to the very treatment (dry boric acid) which had been so extremely successful in a long series of cases, and even then I began to look about for the reason why these cases acted so differently from the usual type of suppuration from the middle ear.

The first comprehensive paper on these perforations is from the hand of Morpurgo,[1] and from it I learned that Orne Green had previously called attention to the obstinacy and the danger of this complicated affection. Since then the number of cases and papers bearing upon the topic has increased so vastly that it would go beyond the limits of this article even to mention them by name. For this reason I will merely suggest, as I proceed, what has particularly exercised any decided influence upon my train of thought.

Some time ago I noticed, as I thought, that the extreme obstinacy of perforations in Shrapnell's membrane in three cases out of seven was due to the presence of epidermic masses near to or close behind the perforation. Since then I have seen over one hundred cases of this particular type, and examined a large number of them anatomically, and I am now convinced that this coincidence is much more frequent than I used to think; indeed I now actually believe that stratified epidermis is constant behind these perforations, and that their extreme frequency in the living has only been demonstrated since the employment of direct expulsion by means of Hartmann's bent-tube tympanic syringe.

Politzer, too,[2] has lately said: "I have most often found this peculiar secretion (greasy clumps, or whitish-yellow shreds, in the syringed-out water) in those obstinate though rather scanty suppurations, with perforation of Shrapnell's membrane, next in cases of adhesion between the *Mt* and

[1] *Arch. f. Ohrenhlkde.*, xix., 1883.
[2] "Lehrbuch," 2te Aufl., p. 320.

the promontory wall, where the secretion can only escape by a minute opening in the posterior superior quadrant of the *Mt*, additionally in cases of protracted suppuration in the antrum, and finally with fistulous openings in the bony meatus."

After observing how differently these chronic suppurations with perforation of Shrapnell's membrane ran along in comparison with the usual type of chronic suppurations of the middle ear, there was no longer any doubt in my mind that in their origin, too, they must differ greatly from the usual forms of suppuration.

My first idea was that the Shrapnell's-membrane cases were nothing but a sequence of the customary type of suppurative middle-ear disease, because the process runs its course inside of the posterior and upper middle-ear spaces separated from the real tympanum, and there leads to secondary perforation of Shrapnell's membrane, lying close alongside, whilst in the tympanum itself the suppuration has long since terminated. This idea seemed to me for a long time unavoidable, because I had never been able to follow in its origin in Shrapnell's membrane, *a fresh perforation in a case of acute middle-ear disease*, whilst a perforation coming on at a late date, and without any aggravated symptoms, might much more often remain withdrawn from our direct observation in a later stage of the middle-ear disease.

Another thing hard to connect with such an origin is the excellent hearing and the relative rarity of cicatrices and perforations in the rest of the *Mt*, together with perforation of Shrapnell's membrane. But the most enigmatical of all the symptoms was the impossibility of ever determining with any accuracy *the precise time at which the perforation occurred.*

Such doubts as these lately induced Walb to advance, in regard to the origin of the form of disease that we are discussing, a new theory, which has proved to everybody as unexpected as it has to me.[1]

He argues that every suppuration in any portion of the middle ear must arise from the access of pyogenic organ-

[1] *Arch. f. Ohrenheilk.*, xxvi., p. 185.

isms. If the ear has previously been intact, the tube alone can serve as an entrance, and the tympanum must have furnished the gate of entry.

Now Walb, like all the rest of us, had never been able to observe an acute suppurative rupture of Shrapnell's membrane, during an extensive suppuration in the middle ear. On the contrary, his experience leads him to think that, in a majority of cases, the infection must have *entered from the external meatus partly in the form of an otitis externa, with ulceration at the margin of Shrapnell's membrane*, partly through a persistent foramen Rivini. He had often additionally witnessed on one side a perforation of Shrapnell's membrane, with suppuration, and on the other side a small aperture at the upper pole of the *Mt.*

I can testify to the fact that such small apertures in the upper margin of Shrapnell's membrane are not at all uncommon. But so long as the existence of a congenital fissure at this spot has not been demonstrated in the newborn in a large number of cases (which in a very large number of sections in normal temporal bones I have never been personally able to discover), we are justified in doubting whether such an aperture as Walb describes may not have been due to a previous suppuration in this region.

The suggestion, however, that an inflammation may extend from the meatus through Shrapnell's membrane into the middle ear, despite the fact that we rarely or never observe such a path, so far as all the rest of the *Mt* is concerned, is well fitted to excite our strictest attention, and to make us extremely careful in adopting the theory of an invariable propagation through the tube, a method of extension which, as we have above remarked, and as Walb has suggested, after careful observations, offers many contradictions.

I must here mention still another observation in cases of perforation of Shrapnell's membrane.

We often find the rest of the *Mt* abnormally concave, with all the characteristics of a long-enduring *closure of the tubes*, an alteration in form that was observed by Morpurgo, and indeed emphasized by him as of great value in a diagnostic point of view, in conjunction with other symptoms.

This concavity I have long considered as the simple result of the perforation in the superior pole of the *Mt*, since we can easily imagine that, owing to the loss of tension which Shrapnell's membrane, although somewhat slack, still exercises upon the short process, the tensor tympani gains the upper hand. Or we might regard this as the expression of more extensive cicatricial synechiæ, which the previous suppuration itself had left behind in the tympanum. It is in this light that this distinct appearance has often been regarded by others besides myself.

Now, on the other hand, observations are constantly accumulating of concavity of the *Mt* and tubal processes where Shrapnell's membrane is perfectly intact. And additionally, when there is no free communication between the upper space and the real tympanic space, we can occasionally prove that the tube is closed on the perforated side, because the hearing is often diminished, but may be increased by using Politzeration.

Walb has seen tubal processes with perforation of Shrapnell's membrane, and I will quote what he says, because it is the first time in literature where these two affections have been observed from the standpoint of mutual interdependence.

"I have many cases on hand in which, in children who suffered from relapses of inflammation of the middle ear, owing to adenoid vegetations in the naso-pharynx, a perforation of Shrapnell's membrane was observed during the early portion of an attack of exudative inflammation of the middle ear, and this, too, after the *Mt* had long been extremely concave, as a result of previous attacks."

Whenever the tubes are permanently obstructed, Shrapnell's membrane, on account of its relaxation, shows the characteristic symptoms of incurvation with great distinctness. Often enough it lies directly upon the neck of the hammer. It is not at all improbable, that slight ruptures occur just at this spot in intercurrent exudative processes.

The only reason why fresh ruptures are not usually observed at this region probably lies in the difficulty of seeing it at all, under the most favorable circumstances. Thus we could explain those frequent though brief discharges from

the ear, observed during the course of occasional tubal obstructions, although no perforation is visible in the entire surface of the rest of the *Mt.* Thus, too, the portions of the shrunken Shrapnell's membrane resting on the neck of the hammer and the upper bony margin could easily adhere to the walls of the small interior space. Every observer must have seen repeated instances of such adhesions.

If we now return to the pathogeny of cholesteatoma, and recall to mind the development of an epidermic layer upon the mucous membrane of the tympanum by the formation of a bridge between the *Mt.* and tympanic wall, we have here in small dimensions in Prussak's space, as well as in the cavity above, defined by Kretschmann, the same procedure as we have easily been able to follow on a large scale in the tympanum of the living.

Here, as well as there, the cutis, by means of its property of extending along the surface can gain the upper hand over the mucous membrane, grow through the small entrance once gained, and extend upon the bridge into the middle-ear spaces laid bare. But when its margins have once become covered with epidermis, the little opening has become persistent.

Then, in the small spaces shut off from the exterior, concentric layers of deposit form as everywhere; the irritation which this causes, excites a still more rapid and renewed formation from the epidermatized wall of the little cavity. Soon it is too small.

The astonishing capacity of swelling epidermis to wear away the surrounding walls, is well enough known from once seeing a case of cholesteatoma. Here the same thing is seen in a smaller space. First, the delicate mucous duplicatures between the hammer and anvil, and the external wall of the Aditus, fall a victim to the increasing epidermic masses, and in the antrum the masses will rapidly extend.[1]

[1] In opposition to generally expressed views, I have considered the projection of the Fallopian canal, the boundary between the tympanum and Aditus, on the one hand, because we thus get the simplest form for these two spaces; for the tympanum, that of a short cylinder, for the Aditus, that of a slightly inclined triangular prism, and between the two lies the narrow space bounded by the bodies of the hammer and anvil. On the other hand, because the morbid processes which go on behind the isthmus and are, in a majority of

Thus the collections of epidermis reach by a short path the space where they have been most often discovered by Virchow and other observers.

I will cite only a single case amongst the large number of pathological specimens upon which this paper is based, a case of cholesteatoma with simultaneous perforation of Shrapnell's membrane, in which the extension of the epidermis through the perforation, and its continuation over the walls of the Aditus, the ossicles, and the antrum, which the growth had secondarily enlarged, are plainly visible. I will also cite only a single clinical case of tubal catarrh, and the immigration of epidermis, and the infectious material from *the external meatus* through a rupture in Shrapnell's membrane.

The patient is a youth of sixteen, who has been under my observation for six years; first applied for advice for right-sided pain, suppuration and disagreeable odor. Perforation in posterior, superior quadrant; the suppuration now and then decreased but never ceased for any length of time. The left ear occasionally suffered from deafness with tubal catarrh, which always rapidly disappeared after Politzeration. Later, adenoid vegetations were snared off or scraped away, but they could not be wholly removed owing to anatomical abnormities of the parts; after removal, the tubal catarrh ceased in violence, though never completely.

Epidermis has often escaped from the perforation during the last few years; the snare was several times employed to remove granulations springing from the tympanum. I observed that when the patient absented himself from treatment for any length of time, the discharge increased in amount and in fetor.

A year ago there was a sudden proliferation at the anterior upper angle corresponding to the situation of the upper pole of the *Mt*, and after this was removed, I discovered a second perforation in Shrapnell's membrane. Injections removed cheesy secretion, clumps of epidermis, and several minute polypi.[1]

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cases, introduced with perforation of Shrapnell's membrane, and cholesteatoma, demand a sharply defined, clinical separation from those in the actual tympanum. This boundary line is indicated from without by the upper marginal tendon of the *Mt.* Schwalbe has lately suggested the facial canal as the boundary between the two spaces. Sexton similarly defines the Aditus (Trans. Am. Otol. Soc., iii., 4), and the space so limited is now called by American otologists generally, the "attic."

[1] Whilst injections with the common syringe are followed by an abundant outflow into the throat, injections with the bent-tube syringe do not run into the throat, but on the contrary, vertigo is always observed.

From this time the fetor and discharge ceased, though there was a slight return last winter during an attack of diphtheritis. Since then the suppuration has gradually decreased without becoming putrid, and in February came to an end after a small coarse polypus was extracted.

My attention has lately been attracted largely to the *left ear*, which I had not looked at for a long time as the hearing remained fair.

The patient returned in April last complaining of discharge and pain in the left ear, cerumen, shreds of epidermis, and discharge in meatus; several days before the syringe could cleanse the meatus, and then a portion of the mass remained fast to the *Mt*, and had to be removed with the forceps.

I now discovered, just above the short process, a point filled with a whitish substance. The margins of perforation pushed forward by the projecting mass; a probe could be passed alongside into the cavity, but neither this nor injections were capable of getting any thing out. Finally, with the forceps I withdrew a small sac, shining like mother-of-pearl, and then a plug the size of a bean, consisting of whitish irregular strata of epidermis.

Since then the slight inflammatory symptoms have terminated, but the dry perforation in Shrapnell's membrane still remains, and gives a good view of the neck and head of the hammer. The probe, pushed far inward and upward, meets with no obstruction. Hearing, right, 4 *m*; left, 5 *m*, and more for whispered voice.

There is hardly any doubt that here we have observed, on the *left side, the same process from its start*, that had already existed in the right ear for years with abundant suppuration. It seems, moreover, that here the pathogeny of perforation in the region of Shrapnell's membrane, as well as the consecutive formation of cholesteatoma is rendered as clear as the day.

The repeated influence of the unilateral pressure upon the *Mt* from without, forces the relaxed and thinned Shrapnell's membrane against the walls of Prussak's space, ruptures it at some spot, and agglutinates it with its underlying tissue. The epidermic covering gradually extending into the small space produces blind sac-like layers (as we were actually able to remove), enlarges in the course of time with successive fresh deposits of epidermis, gradually absorbs the opposite

folds of mucous membrane, and thus boldly and freely advances toward the tegmen tympani into the Aditus.

Whilst the increasing mass acts on the one hand as a foreign body, and on the other hand the layers of the blind sac opening outward and gradually crumbling, favor the entrance of pyogenic or septogenic organisms, and therewith a septic destruction in the interior of the plug, a reactive inflammation is thus produced all around. Just as we so often see granulations at the *outer margins* of the minute perforations, so the same formations originate inward in contact with the plug, and as is the case at the narrow external opening, at the narrowest spot. So long as pyogenic germs cannot enter from without (and this is the case so long as the peripheral layers of cholesteatoma remain intact), these granulations can be transformed into connective and cicatricial tissue. Thus can we explain the frequent occlusion of these morbid deposits from the real tympanum, as Hartmann has lately demonstrated in several post-mortem cases.[1]

If any breach has originated in the epidermic sac, the septic contents in its interior press directly into the exposed Aditus and antrum, and there produce inflammation and ulceration.

A relative cure may follow by gradual extension of the epidermis over the walls of the entire antrum. Still it cannot be a definite one. On the contrary, we find here a territory upon which the processes still in a minor state can repeat themselves on a larger scale, and so gradually wear away the bony walls in all directions.

A few regions where septic deposits have been forced in, such as the ossicles and bony wall of the external perforation, may gradually yield to caries; the external opening gradually becomes larger at the expense of the upper wall of the meatus, till finally those enormous defects which Hummel has observed occur.

I have seen all sorts of transitory conditions between the simple perforation in Shrapnell's membrane, and the entire absence of the upper and posterior wall of the meatus, and I am sure that all of them represent only the beginning and

[1] *Deutsch. med. Wochensch.*, 1888, No. 45.

the end of one and the same morbid process, and to this conclusion I have come despite the extreme difficulty of making a satisfactory examination of this region owing to the narrowness of the space and the foreshortening of the parts when under inspection through the speculum.

Possibly the condition of things that I have here described represents only the pathogeny of one portion of the cases of perforation of Shrapnell's membrane on the one hand, and of the formation of cholesteatoma on the other. Still, for a large number of cases we may take the description of the above case as that of a series of links which gradually lead from simple tubal catarrh to perforation of Shrapnell's membrane, and from this to the formation of cholesteatoma in the Aditus and antrum with all of its frequently fatal complications.

The *treatment* of these cases may occasionally resolve itself into a spontaneous cure. The moment that the cholesteatomatous cavity to any great extent lies bare, whether in the meatus or at the external surface of the mastoid owing to the cicatrization of extensive fistulous openings, the neoplastic formation of epidermis ceases, and the covering acts just like any cicatrix of the skin.

Our task, therefore, can only be either to enlarge the orifice already present by removing the upper bony margin of the perforation in the meatus, or by the extraction of the hammer and anvil, or thirdly, by making a new opening into the antrum. An operative removal of the entire lining membrane of the cavity would not only be improper, but, on account of the complications (the ossicles, if preserved, are invariably covered with epidermis), impossible.

To indicate which of these three ways may be entered upon in any given case, or to decide how far they might be followed up together at the same time, does not lie within the province of this paper.

A CASE OF LEUKÆMIA WITH NOTEWORTHY CHANGES OF THE NASAL MUCOUS MEMBRANE.

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(*With plates i. and ii.*)

LEUKÆMIA is a disease characterized by an increase, either continuous or intermittent, of white blood corpuscles, accompanied by a hyperplasia of the hæmatogenic organs, by hemorrhages, and by heteroplastic formations of lymphatic tissue. Although the disease had probably been observed by Cullen [9] and Duplay [10] an absence of the more delicate modern methods of examination doubtlessly prevented them from understanding the nature of the malady. Nor did Craigie,[8] of whom we might have expected a recognition of the disease, nor Bichat, nor Velpeau, nor even Bennet,[1] come to a correct conclusion as to the nature of the malady, although the last author later on contributed some valuable articles in relation to this disease. Bichat, whose article on leukæmia appeared contemporaneously with Virchow's work on the same subject, explained the disease as pyæmia—suppuration of the blood. Virchow first gave us a correct idea of the malady; we must therefore give him the credit of having really discovered it. Several cases of leukæmia occurring in animals and man have since been published.

[1] From vol. xx., No. 1., German edition, 1889.

In spite of the abundant literature on the subject, the authors do not by any means agree on all points. I hope, therefore, that it will not be out of place to classify briefly the facts already established, and besides to add certain observations of my own made in regard to the symptomatology of this malady.

That leukæmia is an endemic disease cannot be doubted, otherwise the frequency of its occurrence in certain countries, as in the Baltic provinces, and its rare occurrence in other places, Switzerland for instance, could not be explained. Eichhorst,[10a] until 1887, saw only one case of leukæmia in five thousand cases, and the janitor at the pathological institute at Zürich maintains that among the post-mortems made at that place during twenty years only three or four cases of leukæmia had been observed. We shall require fuller statistics before we can decide whether certain tribes or races are inclined to the acquisition of this malady.

Until 1869 the following three forms of leukæmia were recognized: the splenic, the lymphatic, and the mixed form. Neumann subsequently added the myelogenic form. It is as yet doubtful whether Behier's[1a] assumption of an intestinal form is correct. In Behier's case the lymphatic system of the intestines was undoubtedly diseased, and the other organs normal; but nothing is mentioned about the condition of the bones, and a medullary or myelogenic form is therefore not excluded. As to the myelogenic leukæmia of Neumann,[36c] up to 1872 it was generally assumed that the marrow was diseased in every case; for in every case in which the marrow had been examined it was found to have been pathologically altered. And in those cases in which an examination of the bones had been neglected, a myelogenic affection could not of course be excluded. Neumann therefore was certainly justified, in view of his observations[36b] and of the important physiological functions attributed to the marrow, according to his own and Bizozzero's[4a] investigations, to locate the origin of the disease in the marrow, and to consider the changes in the other organs as secondary, and following upon the changes in the marrow. Although the importance of the marrow as a hæmatogenic organ has since

been generally recognized, not only by the investigations of Neumann but also by those of other authors (compare Schoppe [44] and also Englisch,[13] who has found secondary leukæmic conditions of the blood following chronic affection of the bones), a myelogenic form of leukæmia has not been accepted by all pathologists.

In 1872 Ponfick [40] reported a case in which there were no pathogenic changes found in the bones. In 1876 [40b] the same author reports two additional cases by which he proves the existence of a pure splenic form without myelogenic changes, or even pathological changes in the white blood corpuscles. He further concludes that we have no right to diagnose myelogenic leukæmia because of the presence in the blood of corpuscular elements, representing transition-forms. As is well known, cells possessing a colorless nucleus surrounded by a homogeneous yellowishly tinged border were considered by Neumann as characteristic of the myelogenic form. Mosler, on the other hand, assumes a medullary affection when blood corpuscles filled with fatty molecules are present in the blood.

The second case of Ponfick's, in which a marked hyperplasia of the marrow was found, without the presence of transition-forms in the blood, seems rather to support Mosler's second conclusion. Ponfick thinks that the clinician should decide whether in a given case the myelogenic changes are primary or secondary, as it is impossible for the pathologist to decide this question.

Bizozzero and Salvioli also disputed Neumann's [36g] propositions (although the latter still clung to his views until the year 1878), that the nucleated red blood corpuscles found in the blood during life were of great diagnostic value. Fleischer and Penzoldt [16] in the year 1880 also reported a case of leukæmia in which the marrow was not affected, and in 1879 Heuk showed in one case that not only was there an entire absence of any hyperplastic condition of the marrow, but that an atrophy of the marrow due to a general osteosclerosis was even present. Leube and Fleischer,[30] in a marked case of leukæmic condition of the blood, found the spleen, lymphatic system, and marrow to be perfectly normal.

Although the report of such cases tended to weaken the theory that the primary cause of leukæmia was to be attributed to a myelogenic affection, there were still so many cases on record where changes were found in the marrow, and among those cases three of pure myelogenic leukæmia (Englisch,[13] Litten,[29] and Brodowski), that the importance and relation of the medullary changes occurring with this disease cannot be doubted. I refer here only to the publications of Bettelheim,[9] Eisenlohr,[12] Fränkel,[17] Hand,[22] Huber,[25] Immerman,[25a] Fleischer and Penzoldt,[16] Küssner,[26] Lauenstein,[27b] Mosler,[34b, c] Müller,[35] Rusack,[43] Waldeyer,[47] and to my own case. Even Cohnheim, although he did not consider the bone affection as the primary lesion, always found the changes in the marrow described by Neumann. In any case it is remarkable that the origin of the malady should have been imputed to so many different causes, as chronic enteritis, intermittent fever, syphilis, scrofulosis, traumatism, poor hygienic and marked depressive conditions. These of course are all factors, which tend to reduce vitality, and thus enable bacteria to enter the circulation, but we must not forget that in many cases it has been impossible to find any cause for the origin of the disease.

P. Olivier[38] in one case referred the cause to alcoholism; his view, however, has not as yet found any adherents. Mosler[l. c.] considers a traumatic injury of the bones, or the spleen, a certain cause of leukæmia. Although we know many conditions that exist in connection with leukæmia, we as yet have no intimate knowledge of the nature of this malady.

Many theories have been adduced on this subject; they may be divided into two groups:

Some authors claim that an affection of the hæmatogenic organs is the primary cause, and that the changes in the blood are secondary. Others consider the malady to be a primary affection of the blood itself. I will here only refer to the original views of R. Schulz,[45] as I consider that the first-mentioned theory, attributing leukæmia to primary changes in the spleen, glands, and marrow, is generally known.

Schulz considers the lymphatic neoplasms found in leukæmia as proliferations of the mesoblast, representing new formations of connective tissue, as opposed to the atypic epithelial proliferations, carcinoma, for instance. The difference between leukæmia and pseudo-leukæmia would then consist only in the fact that in pseudo-leukæmia the proliferating process is so rapid and so extensive that the lymphatic channels become engorged, and an outflow of the lymphoid cells into the circulation prevented, while in the more chronic condition of leukæmia this engorgement of the lymph-channels does not take place, and therefore the cells are enabled to enter into the blood circulation. I would here only remark that cases of acute leukæmia have been observed without any apparent accompanying engorgement of the lymphatic channels.

Biesiadecki [28] opposes this view of Schulz, and on account of the regressive metamorphoses observed in the leucocytes, considers a primary affection of the blood with secondary changes in the organs as the most probable origin of the disease, especially as we have no absolute proof that a proliferation of white blood corpuscles actually occurs. Löwit and Bizozzero, as well as most observers, now incline to this theory. The leukoblasts of Löwit,[32a] mono-nucleated white cells, containing very little protoplasma and proliferation by a divisio indirecta, per granula, were not found to be present in the hæmatogenic organs (the lymphatic glands and in the marrow). This author assumes that in leukæmia the normal evolution of the mono-nucleated blood corpuscle is arrested, and that therefore the system becomes inundated as it were with these cells. Leube and Fleischer's [30] case shows that an inundation of the organs with lymphoid cells need not necessarily occur. That even an arrest of evolution in the leucocytes may also be absent in leukæmia, has also been proven by some authors. For Bizozzero found several karyokinetic figures in the lymphoid elements of the follicles of the spleen.

Klebs [27] and Ziegler [49] consider the parasitic origin of this malady as not improbable. The former even found lately in a case of acute leukæmia the blood of the patient filled

with short, rapidly moving bacilli, during life as well as after the post-mortem examination of the blood.

I would like to describe shortly, in connection with these remarks upon the ætiology and nature of leukæmia, the changes which have been found in the different organs. The spleen, the lymphatic glands, as well as the other lymphatic organs, the palatal and pharyngeal tonsils, the tonsils of the tongue, follicular apparatus of the thymus, of the thyroid gland, of the conjunctiva, the follicles of the intestines, have been found hyperplastic. In the first stages of leukæmia, the spleen and lymphatic glands are hyperæmic, rich in cells, and soft; they become indurated in the second stage owing to the proliferation of their stroma. In the marrow a similar hyperplasia has been observed. Macroscopically, one can distinguish, according to Neumann, two separate conditions, a pyoidal and a lymphadenoidal change. Under the microscope we observe that the smallest arterial ramifications terminate into large cellular spaces, and that the walls of the arteries are also infiltrated with lymphomatous tissue. Besides these diffuse or circumscript hyperplasias, a heteroplastic condition of those organs which normally have no lymphatic tissue also occurs. Not only have these lymphomata or lymphoid infiltrations been found in the liver and kidney, but also in the pericardium, in the pleura, in the lungs, in the larynx, in the trachea, in the epiglottis, in the pharynx, in the skin, in the epididymis, in the pancreas, in the lachrymal gland, in the posterior portion of the orbital cavity, in the lids, and even in the nerve sheaths. Besides hemorrhages into the different organs, and the presence of degenerative corpuscles in the blood, Liebreich [81a] first described a case of leukæmic retinitis; Pepper observed a hemorrhage in the labyrinth, followed by deafness; Politzer [89] describes changes in the internal ear, accompanied by proliferation of connective tissue; Blau [5] and Gottstein also relate cases of aural disease in connection with leukæmia. I desire to call attention also to the chemical composition of the leukæmic blood, to the quantitative changes in the amount of iron and of hæmoglobin, to the diminution of the specific gravity to 1,040 (1,055 being the normal);

furthermore, to presence of hypoxanthin of lactic, formic, and acetic acid, and finally to the eosinophilic cells found by Ehrlich, in order to have mentioned the most important changes found by the pathologist and chemist.

It is surprising that as yet no attention has been given to the appearance of the nasal mucous membrane; for we surely might expect to find changes in the narrow zone of adenoid tissue and about the glands. I have attempted to supply this deficiency, and will add here the result of my investigation; first, however, giving the anamnesis and the results of the post-mortem examination in a case kindly referred to me by Profs. Klebs and Eichhorst.

Heinrich Fehr, forty-six years old, came into the internal ward of the Zürich Canton Hospital on the 23d of September, 1888. The extreme pallor of the patient, petechiæ in the skin, the enlargement of the spleen and of the lymphatic glands, the condition of the retina—retinitis leukæmica,—and finally the enormous increase of the white blood corpuscles, as well as the diminution of the red (in one cubic centimetre 301,600 red and 306,100 white, found with Thoma-Zeiss' apparatus), permitted no doubt as to the diagnosis, leukæmia. The blood of the patient was of a light-brownish hue, watery; the red blood corpuscles were scarce, pale, and had a wavy border (no thorn-apple shapes being present). The white cells contained only one nucleus, surrounded by a narrow zone of protoplasm. Cells with a yellowish border and colorless in the centre were also found; whether there were transition forms of the red blood corpuscles could not be accurately ascertained. The blood contained only 15 per cent. of the normal amount of hæmoglobin.

The post-mortem showed the following: Skin of pale color, ecchymoses; very little adipose tissue, muscles pale, but otherwise normal. Eyeballs prominent. Spleen 15 *cm* long, 10½ *cm* broad, 5½ *cm* thick, of firm consistency, and of fleshy color, dotted throughout with white spots.

Lymphatic glands strongly hypertrophied, also in places where, as a rule, lymphatic glands are not found; there were marked nodules of lymphomatous tissue present. The glands of the abdomen weighed 1,548 grammes. Left kidney weighed 273 grammes, was 9 *cm* broad, 5 *cm* in depth; the surface, on which several pale-red spots were visible, of white-yellowish color; cortex 7–10

mm in breadth ; the columns of Bertini somewhat enlarged ; the pyramids small, yellow ; the papillæ of pale color.

Right kidney similar to left in appearance and of same weight.

Liver 28 *cm* in breadth ; the right portion 22 *cm*, the left 17 *cm* in height. Surface smooth, of pale-brown color, and also littered with several white spots, especially in its upper portion.

Œsophagus, stomach, intestines, pale, otherwise normal. Lungs, œdematous and hyperæmic. Left apex slightly adherent.

Heart very rich in adipose tissue, and large. The muscles pale, but well developed.

Retina covered with blood extravasations, which were somewhat bulging in the centre. On the right retina the optic nerve surrounded by extravasations in the shape of narrow circular strips.

The marrow of the femur and sternum of gray and of grayish-red color, and very succulent. The marrow cylinders of the femur larger, somewhat reduced in thickness.

The Result of the Microscopic Examination.

In the liver several very small circumscribed and also diffuse lymphoid infiltrations. The single lymph corpuscles had the appearance of small lymphocytes, with a single round nucleus and a very narrow circular zone of protoplasm.

Lymphatic glands almost normal.

In the spleen the follicles were somewhat hypertrophic ; the striated pulpa also thickened. Besides small leucocytes, larger round cells were present, about 0.012 *mm* in diameter, and possessing a slightly bluish-tinted hyaline nucleus.

Kidneys dotted with lymphomata ; the epithelia of the urinary tubules possess a *badly stainable* nucleus. Some of the cells degenerated ; the lumen of the tubuli contorti filled partly with lymph corpuscles and partly with hyaline cylinders. Friedreich, Cornill, and Ranvier found similar conditions. I quote the last-named authors : "Les reins peuvent être le siége de productions secondaires consistant en un tissue adénoide. Ces petites tumeurs ne différent pas de ce qu'elles sont dans tous les autres organes.—En même temps les cellules épithéliales des tubuli subissent la dégénéresçence granulo-graisseux.—La lumière de ces tubes

contient tantôt des amas de cellules lymphatiques tantôt des cylindres hyalins."

The marrow, which for certain reasons I was unable to examine in a fresh state, was examined by placing a small portion in Müller and hardening it in corrosive sublimate. As the celloidin and paraffin sections show the same condition, I will here only give the results obtained in examining the paraffin specimens.

The most prominent feature was the presence of small lymph corpuscles possessing a large nucleus and a very narrow protoplasmic ring. They were embedded in a fine adenoid network. In the specimens that were stained with hæmatoxylin, the red blood corpuscles are easily recognizable on account of their yellow color and are variegated in appearance. Most of them were fully developed—that is, without a nucleus. Several blood corpuscles, round in shape, without nuclei, were also found, similar to cells already described by Hagen as so-called giant blood cells, and found by this author, as well as by Orth and Litten, frequently in the blood of anæmic persons. My attention was further called to some large cells about 0.014 *mm* in diameter, which have been described as occurring in the medulla of patients suffering from chronic diseases, and to other larger roundish hyaline masses, of pale-bluish color, surrounded by a narrow pink crescent-shaped or annular zone, the pink color being due to eosin. On the whole, the medulla showed conditions described by Neumann as the lymphadenoid form of his myelogenic leukæmia.

The other organs were not examined, with exception of the nasal mucous membrane.

The mucosa, both of the respiratory and of the olfactory portion, showed an entirely different coloring from what it possesses ordinarily. Normally the respiratory portion has a pinkish appearance; that of the olfactory region a light-yellow tint, sometimes intermixed with pink. The leukæmic membrane had a moist, glistening appearance, and possessed, throughout its inferior part, up to the middle turbinated, a light yellowish-brown; the upper portion, in-

cluding the olfactory fissure, the superior turbinated, the part below the ethmoidal plate, the corresponding portions of the septum, a dark-brown color. The mucous membrane was also decidedly thickened throughout its whole extent. As I was prevented from examining the membrane in its fresh state, I placed the whole septum in Müller's fluid. The sections thus obtained showed most clearly that lymphomatous nodules were present on the turbinated bodies, on the septum, and especially in the nasal roof.

I was enabled to make teased specimens from the olfactory region and from the inferior turbinated body. The epithelium was well preserved throughout. The ciliated epithelium of the respiratory region as well as of the olfactory region showed nothing abnormal. The epithelium was composed of two distinct groups of cells.

In the first group are ciliated cells having a conical shape and lateral ramifications which mutally interlace—"the supporting cell." The second group embraces the pencil-shaped olfactory cell. Besides these cells already described by M. Schultze,[45b] I found transition-forms between the supporting and olfactory cells, which have been described by others as olfactory rods, olfactory cylinders, and olfactory cones.

The accompanying drawings represent the epithelium and are made from Müller-specimens. The cells marked III *b* and III *bb*, were drawn freehand; the cells III *a 1–31* are exact drawings of the original cells, their outline having been first drawn through a camera lucida.

A second important point, which I wished to elucidate, was the condition of the epithelium in the olfactory tract. Gegenbaur, Leydig, Luschka, Henle, Ecker,[13a] and others, have all observed the vibratory motion of the ciliæ in the olfactory tract immediately after death (in executed criminals). Ecker and Frey, however, in accordance with Schultze's observations, have found in one case that the epithelium of the olfactory tract was not ciliated. M. Schultze maintains that *the olfactory tract in man has no ciliated epithelium.* Man in the course of his development is so frequently afflicted with nasal catarrh, which necessarily affects the olfactory

organ, owing to its somewhat more exposed position, that a normal olfactory tract is very rarely found; hence only portions of the olfactory tract possess a normal olfactory epithelium. If we therefore examine parts of the olfactory tract, which appears normal on macroscopic examination, we will generally find a columnar *ciliated* epithelium, as is the case in infants. It is indeed difficult on account of the frequency of nasal diseases to find perfectly normal olfactory grooves in the adult. In my case I have been able to find traces of ciliæ on the olfactory epithelium.

Although the epithelium was normal, the mucous membrane itself was decidedly changed. The normal condition of the mucous membrane is, as is well known, the following: The numerous tubular glands of Bowman are situated immediately beneath the epithelium; the intertubular connective tissue is scarce; the adenoid tissue, which is present in the respiratory tract, is entirely absent in the olfactory portion. In our case only remnants of tubules were found, filled with degenerated cells, the nuclei of which absorbed the staining fluid poorly, and without pigment. The subepithelial layer was enlarged, replaced by a lymphoid tissue, which was present either diffusely, or in circumscribed distinctly separate, ovoid, or round groups. In the centre of these circumscribed miliary lymphomata, I usually found a tubule, a nerve filament, or a blood-vessel. Here and there also yellowish-brown pigment. Whether this pigment was composed of hæmatoidin or hæmosiderin, I could not decide in my Müller-specimens, as only alcohol specimens would permit a micro-chemical test.

The grouping of the lymphatic tissue around remnants of glands, around nerves, and blood-vessels, corresponds exactly to the conditions found in the mucous membrane of the respiratory organs by Virchow, and to the leukæmic formations in the kidney, where the lymphatic tissue usually forms around the urinary tubule and, if I may use the word, suffocates them.

The blood-vessels contain many white blood corpuscles; the walls of the arterioles also were frequently found infiltrated. The normal adenoid zone of the respiratory tract was

greatly thickened, to about three times the normal thickness. The tubular glands of this tract were also degenerated. A great deal of pigment was found on the mucous membrane, especially right under the epithelial layer and in the neighborhood of the blood-vessels. This sufficiently explains the yellow appearance of the whole membrane.

The "swell-bodies" of the middle turbinated did not vary any from the normal, except that the venous plexuses contained many white blood corpuscles. There were no hemorrhages found in the parts that I examined.

In this case of leukæmia we have found a perfectly normal condition of the olfactory epithelium, accompanied by a total destruction of the glandular apparatus. This observation can be judiciously used to determine whether the Bowman glands are necessary for the perception of smell. Further clinical and anatomical investigations will perhaps decide this point.

I regret that no examination of the sense of smell was made during the lifetime of the patient, nor did I succeed in obtaining any information through the anamnesis.

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ANATOMICAL DESCRIPTION OF THE LEFT EAR OF A MAN WHO DIED OF MENINGITIS.

By Dr. H. STEINBRUEGGE, of Giessen.

Translated by Dr. J. A. Spalding, Portland, Me.

I REPORTED in the fifteenth volume of these Archives the post-mortem description of the ears of a patient who died of cerebro-spinal meningitis. Three days before he died, the present patient entered the hospital with the same disease.

In childhood had measles; six years ago pneumonia, but otherwise healthy and strong; age twenty-eight. Two years ago he fell to the ground from a second-story window, injuring his left temple, and inflicting a compound fracture of the left forearm. He lay unconscious for three weeks; when he recovered, the sight of his left eye was impaired, and now it is totally blind; forearm useless.

May 20th.—Whilst apparently as well as ever, his head began to ache suddenly, fever and vomiting ensued; unconsciousness rapidly supervened, and at night he was brought to the hospital. He remained in deep somnolence all night, though occasionally complaining of pain in his head; face very red, and near the temple a non-adherent cicatrix an inch in length. Excessive double myosis, slight conjunctival injection, left globe slightly prominent. Ptosis left side and slight divergent strabismus; reflex iridoplegia; tongue slightly coated; strong carotid pulsation; respiration 80, pulse 88.

Morphia, ice-bags; restlessness persisted through the next two days; tossing to and fro all the time. May 23d, torticollis; May 25th, screaming, and pain in back up and down; May 26th, left optic atrophy (not recent).

June 8th.—Patient leaves his bed; relapse June 14th, with headache and vomiting; then gradual recovery.

Both *Mt* dull; H, right ear, watch $\frac{0.05}{10.00}$; left, $\frac{c}{10.00}$. Bone-conduction, R, from temple and mastoid; no bone-conduction in the left ear. Rinne's test positive on both sides. Voice, R, $\frac{4}{20}$; L, $\frac{1\text{f}}{20}$.

The tinnitus rose and fell in coincidence with the exacerbations of the disease. Bone-conduction was only once better for the left ear.

June 10th.—No vertigo or staggering.

June 17th.—Relapse with headache, vomiting, whistling in left ear, and decreased hearing.

June 18th.—Walks about feeling well, but within a week headache, vomiting, and tinnitus.

July 3d.—Whistling in left ear has ceased; watch 0.20, L, and 0.40, R; tuning-fork a' rather better by bone-conduction right.

The patient had never observed any loss of hearing before this illness.

I often saw the patient on the street during the next two years, and he always reported his hearing and his health to be good. About Christmas I heard to my surprise that he had succumbed to a second and brief attack of meningitis.

I append a brief history of the fatal attack:

December 10th.—Violent pain in occiput and temple; loss of consciousness, with occasional attacks of delirium.

December 12th.—Breathing irregular, stertorous; right pupil only as large as the head of a pin, left rather larger; right reacts well to right, left only consensually. No lack of symmetry in the face. Reflex excitability of lower extremities very much increased. Temp. 39.8° (102° F.), pulse 100. Diagnosis: meningitis; calomel 0.25 three times; ice-bags to occiput.

December 13th.—About 11 A.M. sudden injection of right conjunctiva, and œdema of right eyelids. At noon temp. 41.3° (106° F.), and at 3.30 P.M. sudden cessation of respiration.

POST-MORTEM EXAMINATION.

Calvarium symmetrical, normal thickness; diploë moderately filled with blood; bag of pus in anterior cup of skull. Beneath this an irregular line denoting the locality of a cicatrized fracture; at the front end of this line two bits of bone that had been split

off but healed again. Further in front a defect in the orbital roof 12 *mm* in length. At the base of the left temporal lobe a flattening $3\frac{1}{2}$ *cm* long and 4 *cm* wide, with yellowish thick and hard tissue at the margins. At the right temporal lobe a coarse patch $1\frac{1}{2}$ *cm* in diameter. Sections here show the gray rind and a portion of the white substance entirely destroyed, and on the left side yellowish sclerosed tissue containing a small abscess. Membranes at base of brain infiltrated with pus around the chiasma. The membranes of the left hemispheres are noticeably infiltrated with pus. Lateral ventricles greatly dilated, full of abundant fluid, and containing clumps of pus; central ganglia normal. Brain substance soft, very bloody, and shiny.

The fracture had extended into the left foramen opticum, and the compression there had atrophied the optic nerve.

CONDITION OF THE LEFT EAR.

External and Middle Ear.

Meatus wide, *Mt* gray and dull, mucous membrane thickened, bone massive, mastoid process with medium pneumatic spaces, the rest is spongy. Tegmen tympani thickened, ossicles delicate and movable, no signs of adhesive processes, tympanic mucous membrane almost normal, though its violet tinge and the injection of some vessels indicate venous hyperæmia. Bony tube narrow.

MICROSCOPIC EXAMINATIONS.

Nerves.

Auditory nerve surrounded with fresh pus, near the modiolus a defect in the main trunk filled with pus corpuscles; within the latter mass sections of blood-vessels, connective-tissue fibres, and slight traces of the nerve. It is only in the bony modiolus that the nerve fibres begin to appear, though even then surrounded with pus corpuscles. Ganglia cells in Rosenthal's canal slightly altered, as well as the nerves within the lamina ossea spiralis.

Nerve of vestibule surrounded with pus cells; the ampullar branch shows incipient signs of decay.

Pus extends along the facial nerve to the ganglion geniculi.

Vessels.

Veins of modiolus congested, ruptured, occasionally surrounded with brownish pigment; arteries empty.

Convolutions of the Cochlea.

No coarse destruction ; well-marked contrast between recent and old invasions of the morbid poison ; to the *recent* belonged the fresh pus corpuscles at the margins of the scala, on the crista spiralis, and on the tympanal side of Reissner's membrane ; near the latter scattered about as thickened, amorphous, cheesy, brownish masses ; chiefly, in the scala tympani, at the first convolution below the lamina spiralis, and on the inner surface of the fenestra rotunda ; connective tissue at entrance to vestibule. These were all evidently remains of *previous* inflammatory products.

There was no pus in the orifice of the aquæductus cochlea or canal, though possibly its lumen had, at some spot since the previous inflammation, been either closed or contracted. When the section cuts the canal obliquely, it is very difficult to investigate or to discover any obliterated spots.

The condition of Reissner's membrane in the middle convolution was quite remarkable ; thicker, with yellow pigment, and partly agglutinated with the crista as well as with Corti's membrane. It was only at the free end of the latter that it arose high enough to cross over the ligamentum spirale ; the ductus cochlearis was correspondingly contracted near these alterations. These were probably *early* alterations.

Corti's organ preserved ; external cells occasionally absent or ruined, the whole organ darkened with coagulated endolymph, but whether due to the decalcination method or to pathological alterations I cannot decide, and all the more since it would be difficult to ascribe them to the meningitis, which, according to my belief, chiefly attacks the endosteal lining membrane of the perilymphatic spaces. We should then be compelled to believe that the morbid virus had reached the ductus cochlearis through the zona perforata, and was capable here of exciting inflammatory processes, then, capable of spontaneous retrogression and after years leaving only indistinct traces of its march and presence.

Vestibular Structures, Ampullæ, and Semicircular Canals.

Sacculus shrunken, its neuro-epithelium indistinct, its external walls collapsed, and the small interior space filled with coagulated endolymph.

No coarse alterations in the utriculus, though the sections were far from successful ; ampullæ and canals, no coarse alterations ;

cupulæ preserved, a sign that there could not have been either recent or former alterations in the nerve epithelium to any extent. Membranous canals and connections, no abnormities.

REMARKS.

This case deserves especial notice in a clinical point of view, because the patient suffered from three attacks of meningo-encephalitis; the first of traumatic origin, as can be proved by the confinement to the bed, with loss of consciousness after a fracture of the skull, though exact data are absent.

The second attack was possibly due to the epidemic of cerebro-spinal meningitis then prevalent here and there in the country; this may be proved by the violent pain in the back and neck, the long duration, the two relapses, and the herpetic eruption. The case is also interesting as testifying to the claim that persons with previous injuries of the skull are especially susceptible to the virus of cerebro-spinal meningitis. Thus Jaffé [1] reports a fatal case of this disease in a man who had suffered from an injury to the skull from falling from a ladder, and expresses the opinion that the injury had accidentally coincided with a previous infection from the virus of meningitis, though really just the opposite occurred. The author cites a paper by Berger [2] of two cases of cerebro-spinal meningitis following injuries to the head; both of these recovered. Further, Jaffé quotes a remark of Leyden's [3] to the effect that slightly wounded people show great predisposition for the disease whenever a prevailing epidemic offers them a favorable opportunity.

Curschmann received this suggestion with some reserve at the Hamburg Medical Society in July, 1885, because so many patients unreasonably referred almost any disease to any previous injury, no matter how slight; and furthermore, because patients who had been accidentally injured during the initial stage seemed to show about the same set of symptoms as the average run of cases.

[1] *Deutsch. Arch. klin. Med.*, xxx., p. 332.
[2] *Deutsch. Zeitsch. f. Chirurg.*, xii., p. 509.
[3] *Klinik. d. Rueckenmarkskrankheiten*, i., p. 426.

Our case seems to bear out Leyden's opinion.

We shall have to leave it undecided whether the cerebro-spinal meningitis was produced by the virus of a common meningitis or by that of the so-called epidemic cerebro-spinal meningitis, whether it came from without or had remained latent for a time within the skull. The first question belongs to the province of the bacteriologist, and could only be solved if the germs of the two morbid processes were better known, that is to say, the poisonous matter that they produce. It is, at all events, worth noticing that, in the latter attack of meningitis also, the disease extended into the temporal bone along the nerve sheaths of the auditory and facial nerves, and produced rapid destruction of a portion of the substance of the auditory nerve. If this can happen in the ordinary form of meningitis, the question then would be whether disturbances of hearing which, in injuries of the skull, are generally ascribed to a concussion of the labyrinth or to a fracture of the temporal bone, cannot also occasionally depend upon meningitis.

It is to be regretted, so far as the otological value of the case is concerned, that the patient died from a relapse of the meningitis, and not from some other disease, because in the latter case we should be better able to estimate the value of the remnants of the previous ear disease, particularly the condition of the nerve trunk. There is some doubt whether the defect in the nerve, before its entrance into the modiolus, is of recent or ancient origin, and whether the fissures (if we agree that the case is old) were only filled with freshly-formed pus. This view is opposed, as I believe, by the relative improvement and preservation of the hearing in the latter years of the patient's life, whilst total interruption of nerve conduction must have resulted in total deafness on the left side. For this reason the venom of the infection which killed the patient inside of three days must have equally been the reason why the auditory nerve substance was so rapidly destroyed. And then we meet again with the unexplained fact that the fibres of the facial nerve, although equally exhibiting traces of contagious material, offered still greater resistance. There is no mention, in the history of

the last illness, of any paralysis or spasms of the muscles of the face.

The last attack of the disease consisted chiefly of an invasion of the labyrinth along the nerve fibres, whilst the aquæductus cochleæ remained intact. Neither was there any necrosis in the labyrinth similar to that observed in cases previously reported by Habermann and myself. This escape I should like to attribute to the fact that the virus of the epidemic of 1885, which allowed a large majority of the patients to recover, might have been less malignant than usual, just as in other infectious diseases we see many variations between the almost instantaneous death of the tissue and the slightest inflammation. The escape of the labyrinthine tissues from involvement in the last attack of the destructive process, and the limitation of the latter to a portion of the nerve fibres, may be ascribed to the fact that death ensued before the virus could exercise its deleterious action upon the endosteum of the labyrinthine spaces, and perhaps also because its more rapid extension through the aquæductus cochleæ was for some reason obstructed.

No results of the first labyrinthine affection were visible, except the traces of pus in the first cochlear convolution, the partial adhesions of Reissner's membrane, and the doubtful state of things in Corti's organs and in the sacculus.

We should not forget that the patient did not suffer with vertigo, or show the least stagger in his walk, with which fact the normal condition of the semicircular canals and the ampullæ curiously coincides.

So far as the tests for hearing are concerned, it might be questioned whether the hearing had not already been reduced by the injury and associated cerebral disease, and whether the opacification of the *Mt* indicated chronic inflammatory processes in the tympanum and *Mt.* The patient could scarcely have had entirely normal hearing, and yet he assured us that before the injury he used to hear perfectly well, so that at all events the loss of hearing could not have been very great. Additionally, the post-mortem examination failed to show any decided alterations in the tympanic apparatus. This much is plain—that the subjective

noises were not perceived until the meningitis was well under way, and that as convalescence proceeded the noises as well as the hearing spontaneously improved, all local treatment being omitted from the start.

Then again these tests were hardly reliable, owing to the extreme difficulty of examining a patient, who could not easily be moved about in his bed on account of pain, and who, additionally, was not very thankful for any examination of his ears or hearing. Then when the patient began to recover, and as there seemed no chances of proving anatomically the exact condition of affairs, and further still, as it was difficult to tell how far the accident to the head may have affected the hearing, no more searching tests of hearing were made. Nevertheless, the examination revealed some interesting points in regard to the perception of tuning forks.

In this case there was no doubt of a primary labyrinthine disease. Therefore the diminution of bone-conduction for the watch as well as for the fork, to whose importance in labyrinthine diseases Politzer particularly refers, is of great interest. The watch was not heard by bone-conduction in the worse affected ear, neither from the forehead nor from the mastoid process. It was not until the fifth week of the meningitis that the watch could be heard again by bone-conduction. So, too, the duration of perception for the fork by bone-conduction was diminished, for, despite the simultaneously diminished aërial conduction, Rinne's experiment resulted negatively. Weber's test gave various results; the patient heard the $a^{\sharp}$ fork from the forehead, at one time in both ears alike, at another time louder in the right ear, and once only louder in the left ear. These variations may have occurred many times, yet only once at the precise time of the examination.

If, now, relying upon the majority of previous post-mortem examinations, we can assume that the virus of cerebro-spinal meningitis extends through the aquæductus cochleæ and into the trunk and ramifications of the auditory nerve in the labyrinth, we may rationally conclude that *the nerve itself* in these cases is functionally affected. And in our

case it is easy to believe that the diminution of hearing could be referred chiefly to disturbances in the nerve, for neither in the liquid nor solid portions of the sound-conducting apparatus were any obstructions to the conduction of sound anatomically discoverable. Therefore, the nerve conduction was at least partially disturbed, whilst another portion of the fibres must have been in a state of irritation, which could rationally be concluded from the complaints of roaring and whistling in the left ear. The intensity of the subjective sensations of sound varied in direct proportion to the violence of the pain in the head, and must consequently have been influenced by congestive conditions. To this combination of paralysis and irritation in the auditory nerve, and the variations in the latter, I should like to refer the alterations in the tuning-fork perception in Weber's experiment in this case. I call particular attention to the fact that in these cases we say nothing of a physical increase or diminution of the bone-conduction, and interpret Weber's experiment inversely, as in diseases of the middle ear, because the clinical observation indicates a disease of the nerve. If we had not known the nature of the disease, we should, with the usual interpretation of Weber's experiment, have been compelled to regard, at one time the right ear and at another the left ear, as the worse of the two, although the left was the worse at the start and remained so to the end. Then, too, the result of Rinne's experiment is in this case most easily explained by preponderance of the paralytic condition in the nerve, so that the bone-conduction for the tuning-fork became weakened. Possibly, too, the variations in the excitation and the occasional preponderance of the irritative condition could have been proved as well by Rinne's experiment if it had been oftener employed. It is worth observing in this respect that when this experiment was tested, at the time when the hearing was first examined, no subjective sensations of sound had then been complained of.

If, then, the auditory nerve is simultaneously affected in acute diseases of the middle ear (and in the very severe cases it must be granted, considering the congestion of the entire

organ of hearing), I believe that the result of any test with the tuning-fork must be, to say the least, modified by the condition of the nerve. But if it is a question of distinct complications between diseases of the middle and the inner ear, in acute or chronic cases such a modification is quite unavoidable, only it is much more difficult to judge than in case of a simple limitation in diseases of the middle and the inner ear.

THE AFFECTIONS OF THE EAR IN TABES DORSALIS.

By Dr. LEOPOLD TREITEL, Berlin.

Translated by Dr. Ward A. Holden, New York.

FROM the fact that so little attention has been paid to the affections of the ear in a disease which has been studied from such varied standpoints as tabes, we may conclude *à priori* that aural affections are not frequent symptoms of tabes.

The statements of the authors who have written exhaustively on tabes, rest rather on their experience in general than on the results of special examination of the ear. All agree, however, in saying that disturbances of hearing are rarely found in tabes. Charcot[1] states that he can find no explanation for the fact that in tabes affections of the auditory nerve are so much rarer than affections of the optic nerve. Erb,[2] who has treated this subject more exhaustively than any other author, grants that deafness is occasionally found, but he believes that for the most part these cases show merely the simultaneous occurrence of two frequent diseases. While on the one hand he speaks of two necropsies of Lucae, in which the changes were found chiefly in the middle ear, the nerve being intact; on the other hand he gives a clinical observation of Moos, which he believes himself justified in considering a pure or chiefly nervous deafness, analogous to the atrophy of the optic nerve. "However," so closes the chapter, "the question of the occurrence of affections of the auditory nerve in tabes demands a more accurate investigation." Leyden,[3] who also refers to

Lucae's two cases, makes, in his "Clinic of diseases of the spinal cord," the mere statement that he had observed deafness a number of times in tabes, while, in treating of this subject in Eulenburg's "Real-Encyclopædie,"[4] he speaks of the rarity of its occurrence.

He has observed that ear troubles affect especially those cases which have begun in the cerebrum, and those as well which are complicated with amblyopia. Moos,[5] in his investigations on the combined occurrence of disturbances of the ear and the eye, comes to the opposite conclusion; he was rarely able to meet the two together. While I pass over several unimportant publications, I shall mention the clinical observations of Voigt,[6] who, among one hundred tabetic patients, found aural affections twice only, which he concluded unwarrantedly to be affections of the nerve, since a special examination discovered no other aural disease. Up to the present time there have been in tabes no systematic examinations of the ear, according to the principles which are now used for diagnosis in otology.

It seemed to me best to study, for the most part, clinic patients, since these being often examined are better able to give precise statements, and besides this, there is always the possibility of being able to verify at the necropsy the uncertain clinical diagnosis. The cases of anatomical investigation on the changes in the ears of deaf tabic patients have so far lacked special examination *in vivo*. This is to be regretted most in the cases of Strümpell and Oppenheim-Siemerling, since they are, to my knowledge, the only ones in which histological changes have been found in the auditory nerve. In Strümpell's[7] case there was a peripheral neuritis, which apparently did not depend on a preceding meningitis. In the case of Oppenheim-Siemerling there was found atrophy of a large number of those fibres which spring from the nucleus near the vagus nucleus and pass over the ascending root of the fifth nerve. Through the kindness of Dr. Oppenheim I was enabled, in the course of a few months, to examine thoroughly ten cases in the nervous department of the Charité; these were of a more or less advanced tabes. Besides these, I have studied the ears of ten

unselected policlinic cases of tabes, partly in the ambulatorium of the Charité, partly in that of Professors Mendel and Eulenburg. I shall not give full histories of these twenty cases, since I should only repeat, for the most part, the ordinary symptoms of tabes, but shall limit myself, after explaining the principles of my investigations, to recording the results, and to describing in detail only those cases of special importance for our subject.

As to the methods of examination, after viewing the membrana tympani, I tested its sensibility and that of the external canal, then that of the fifth nerve in general. The hearing power was tested by means of whispered words, and where these could not be heard, by words spoken aloud, and that ear was considered normal which understood all the chosen whispered words at ten metres' distance. In the cases of deafness, the catheter was used, and the power of hearing tested a second time. Then followed the tuning-fork test; a *c* fork (an English fork, provided with clamps, introduced into Germany by Lucae) and an $f^{\sharp iv}$ fork were used. On both forks there was an arrangement by which when a button was pressed a hammer fell on the fork, and was then thrown back by a spring. The blow was therefore absolutely regular, and the duration of the vibration, estimated for the ear of the investigator, was almost constant. This period of duration was for the *c* fork per os 12 and further 15 seconds in the air; for the $f^{\sharp iv}$ in the wards of the Charité, 45 seconds; in the ambulatorium of the Royal Ear Clinic, 50 seconds (this difference for the $f^{\sharp iv}$ fork is to be explained by the presence of the beds in the wards, which act as a damper to the sound).

The Rinne experiment was made with the *c* fork in every case; the Weber experiment was made only in special cases. With the $f^{\sharp iv}$ fork the time was estimated only for air conduction, the fork being gradually brought nearer the ear from an initial distance of 0.5 *m*, since the intense sound in the beginning soon deafens the nerve. [6]With the fork tests I believe, with Lucae[9] and others, that a diagnosis of nervous deafness, *i.e.*, an affection of the auditory nerve trunk or its terminations in the labyrinth, is justifiable only where, with

a lessened hearing distance for the voice and a decreased bone-conduction for the watch and fork, the perception of tones of high pitch, as a representative of which I have chosen $f^{\sharp iv}$ is especially diminished in comparison with that for grave tones. It goes without saying that there may still be an affection of the conducting apparatus, "but the better grave tuning-fork tones are recognized by the deaf ear, the more surely may disease of the conducting apparatus be excluded," although this is not the only criterion for disease of the middle ear. A differential diagnosis between an affection of the nerve-trunk and an affection of its terminations, which Gradenigo[10] endeavors to make, is not at this time possible.

Besides this, I have tested in seven clinic patients the electric sensibility of the auditory nerve by means of an oblong plate of 40 *sq. cm* area placed on the cervical vertebræ and a circular plate of 4 *cm* diameter placed just in front of the tragus.

The results were as follows: A disturbance in the hearing could only be produced in five cases; in one case subjective noises were heard, although examination showed no alteration in the ear. I give here the histories of these five cases as far as they are of interest.

Case 1.—Adolf Wehrfeld, æt. fifty-four, was received at the Charité Sept. 24, 1884. In 1866 he had a stroke of apoplexy, which left no consequences, but from about that time he has been somewhat hard of hearing. Within this period came on lancinating pains in the lower extremities, disturbances of vision, loss of taste, and for a year and a half before his admission to the hospital, giddiness when the eyes were closed, girdle sensations, and paræsthesia of the feet. The entry for Sept., 1884, has: mind clear; left eye nystagmus, pupil irresponsive, right incomplete abducens, almost complete oculomotor paralysis; all twigs of the left facial paretic; considerable disturbance of sensibility. Knee-jerk wanting. Hardness of hearing (without accurate determination). Incontinentia urinæ, ischuria paradoxa.

March, 1889.—Patient is so deaf that loud conversation is heard only near the ear. He hears better with the right ear than with the left. Pain-perception is dulled in the region supplied by the

fifth nerve ; pin-pricks produce the proper feeling of pain only in the occipital region (a note to this effect was entered in the journal June 6, 1885). The patient states that for two or three years the teeth have fallen out painlessly. Salivation exists.

The hardness of hearing had come on gradually since 1866 ; since 1883 the patient had been unable to hear the telephone, since 1886 had not been able to understand ordinary conversation.

The left ear had always been worse than the right. Since 1883 there had been subjective sensation of noises, especially the sound of bells, which came on at intervals often of a half year, and lasted eight days or longer, becoming at times so distinct that the patient would get up at night and open the window in order to find whether the sounds did not come from without. The patient has no hereditary history of aural disease ; personally he had had nothing of that sort before 1866.

May, 1889.—Right : membrana tympani so opaque that it has the appearance of milk-glass, yet with no evidence of retraction ; in the anterior half at 3, if one suppose the membrane numbered like the dial of a watch, there is a chalk mass as large as a pin-head. Loud conversation is heard at 0.4 *m.* Catheterization produces a broad, dry sound, but does not improve the hearing. The sensibility of the membrana tympani and the auditory canal, on being touched with the sound, is markedly diminished ; the entrance of air in catheterization is not perceived.

Fork *c* is heard per os 6 seconds, 16 seconds longer in the air. On the vertex it is heard equally well in the two ears, but not so long as on the mastoid process (3 seconds). The patient differentiates between the tone and the feeling of vibration, which lasts longer than the tone-sensation ; $f^{\sharp iv}$ fork heard 5 seconds (normal 45 seconds). The watch can be heard neither per os nor in the air.

Left : membrana tympani opaque, especially in its posterior portion, but not so markedly so as the right ; no evidence of retraction. Loud conversation is understood at 0.15 *m.* Catheterization gives a broad, sharp sound, but does not improve the hearing. Sensibility of the membrana tympani and canal lessened, but not so much as on the right side ; the entrance of the air in catheterization is perceived by this ear.

Tuning-forks : *c* is heard per os 4 seconds (the vibration is felt 8 seconds longer), further 8 seconds in the air, *i.e.*, Rinne + 8. (The simple statement Rinne + 8 is not sufficient, since the de-

gree of the positive result of the experiment depends in reality on the duration of tone-perception through the bone.)

The watch is heard neither per os nor in the air.

Aug., 1889.—Condition as before.

Sept., 1889.—Electric examination, right: Patient hears a hissing sound "as of water boiling," with 0.25 milliampère with cathode closure and cathode duration, not with cathode opening and anode closure, but with anode opening, and indeed it outlasts the anode opening, but stops at once with anode closure; with 0.5 milliampère with anode closure and only with 1.5 milliampères with cathode closure.

Left ear examined after the right; with 0.25 milliampère no reaction, with 0.5 milliampère paradoxical reaction; sensation of sound in right ear with anode opening, when the experiment was repeated, also in left ear, again only in right; later the sound-sensation appeared with anode opening with 0.25 milliampère and lasted beyond the opening of the connection, to vanish again with its closure. With 0.5 milliampère sound-sensation with cathode closure, with cathode duration and with anode closure. With 3 milliampères no cathode-opening reaction is obtained; the examination was not continued further, since a metallic taste in the tongue was noticed.

Case 2 (Policlinic).—Mrs. Schliepmann, æt. forty-eight. Advanced case of tabes, with affection of the cerebrum. Half a year previous patient had a stroke of apoplexy, from which a one-sided paresis has remained.

Patient has had hardness of hearing for three years; no hereditary history of aural trouble. The hardness of hearing has developed gradually. The patient was catheterized for some time by Dr. Hartmann without improvement. She has often a feeling of fulness in the ears, but this is not constant; occasionally she has attacks of giddiness, but without vomiting.

Examination.—Membrana tympani on both sides considerably opaque, with retraction. Sensibility not lessened. Whisper heard both sides at 0.5 *m*. Catheterization, which gives a broad, sharp sound, improves the hearing of the right to 0.8 *m*, the left not at all.

Rinne *c* positive, yet shortened both sides; per os 5, and further 8 seconds in the air; on the vertex the tone is not heard, only the vibration felt, and this lasts but a few seconds.

$f^{\sharp iv}$ right 12, left 15 seconds (normal 50 seconds).

Watch not heard per os, in the air only when very near the ear.

CASE 3 (Policlinic).—Mrs. Peter, æt. thirty-five. Tabes for ten years; had ear trouble when a child; has at present no aural affection.

Examination.—Membrana tympani: right, normal; left, cicatrix, with chalk deposits about it. Whisper heard—right at the normal distance, left at 4 *m.*

Rinne positive: per os, on both sides, 12 seconds; in the air, right, 8 seconds longer; left, 2 seconds longer. The sensibility of the right frontal protuberance is much lessened in comparison with that of the left; a difference in the duration of the tone-sensation is not found. $f^{\sharp iv}$ both sides, 35 seconds.

CASE 4.—Miss Lau, æt. thirty-four, received into the Charité in August, 1889. Advanced tabes, with atrophy of the optic nerves; began seven years before. Psychic disturbances; failure of memory, slight excitability. In childhood the ears had discharged. Since 1885 she has had repeated attacks of giddiness, with vomiting.

Examination.—Right: membrana tympani retracted, cicatrix in posterior inferior quadrant, left, large cicatrix in the anterior half, with extensive chalk formation in its neighborhood. Sensibility of the membrana tympani normal on both sides. Whisper heard both sides at 8 *m.*

Rinne c: right + 8, left + 6 seconds (bone-conduction, 12 seconds). $f^{\sharp iv}$, both sides, 30 seconds. The tone is heard with pain. The electric test, up to 3.5 milliampères, gives no reaction, and was not increased on account of the occurrence of symptoms on the part of the cerebrum.

CASE 5.—H., officer (Policlinic). Since his military service (artillery) the hearing is poor.

Membrana tympani, both sides, retracted and opaque; sensibility a little lessened.

Whisper heard: right at 7 *m*, left at 3.5 *m.*

Rinne *c*, both sides, + 10 (per os, 12 seconds). $f^{\sharp iv}$, both sides, 40 seconds.

I shall give besides these the following history, since it offers much that is of interest.

CASE 6.—Ed. Krüger, æt. forty-nine. Tabes, with disease of the lateral columns. Amaurosis. Since September, 1888, in the Charité. In the region supplied by the fifth pair, pain-perception

is diminished, right more than left. History negative as regards hearing.

Examination.—Membrana tympani : right, opaque in its periphery ; left, total membrane chalky, excepting an oval cicatrix behind the malleus. Right, sensibility considerably lessened ; left, slightly lessened. The patient states that he often feels a drawing in the right half of the face, always accompanied by ringing in the right ear.

Hearing distance for whisper, both sides, normal with the fork.

Electrically no reaction of the auditory nerve is produced by 14 milliampères.

Among the five cases of hardness of hearing there are three in which the disturbance of hearing is inconsiderable, and it may be considered undoubtedly an accidental complication. In but two cases (Wehrfeld and Schliepmann) can the deafness be considered, according to the principles laid down above, to be probably due to an affection of the nerve, since in both, the hearing distance for speech, the bone-conduction for the tones of the fork and watch, and the perception of acute tones are markedly lessened.* Nevertheless careful study of these cases makes it seem to me probable that not only the sound-perceiving apparatus but also the sound-conducting apparatus is diseased.

In the case Schliepmann the marked shortening of the result of Rinne's experiment, although this was positive, the sharp, broad auscultation sound in catheterization and the slight improvement that this latter caused, as well as the opacity of the membrana tympani, would indicate that besides the nerve affection changes have occurred in the middle ear which from the lack of retraction of the membrana tympani can with tolerable certainty be considered sclerotic.

In the case Wehrfeld the marked changes in the membrana tympani and the shortening of Rinne's experiment point to changes in the mucous membrane of the tympanic cavity, which depend on a retrograde metamorphosis, and may also be called sclerosis. I believed in the beginning, since the case Wehrfeld was the first that I studied, that the sclerotic,

* Typical symptoms of Ménière's disease were found neither in these nor in the other cases.

or, better, the trophic disturbances in the mucous membrane of the tympanic cavity were caused by the anæsthesia of the fifth nerve, since in this case a marked anæsthesia of the membrana tympani and mucous membrane of the cavity existed; further studies taught me, however, that this connection was not a causative one. Since on the one hand there were cases, *e.g.*, No. 6, in which a lessening of sensation was found without disturbance of hearing; on the other hand the case Schliepmann showed a disturbance of hearing analagous to that of the case Wehrfeld, without a lessened sensibility. The conclusion follows that particular trophic fibres must also be diseased in order that these changes may occur in the mucous membrane of the tympanic cavity, and that these may be affected alone or at the same time with the sensory fibres.

Aside from the very frequent trophic changes in tabes in general, this conclusion is supported by the fact that in the case Wehrfeld trophic disturbances in the region supplied by the fifth nerve—painless falling out of the teeth,—and probably also of the glosso-pharyngeal nerve—the obstinate salivation,—were found, two nerves which send twigs to the mucous membrane of the tympanic cavity. Further, it seemed that in several tabetic patients the membrana tympani showed an opaqueness not corresponding to the age of the patient. It may be possible that these changes in the mucous membrane only lead to disturbances of hearing when they affect the articulations of the ossicles or the coverings of the foramina ovale and rotundum. I am then inclined to believe that *sclerosis of the posterior columns of the cord not rarely causes trophic changes in the middle ear, which may lead to disturbances of hearing.* I find, however, in the comprehensive account of trophic disturbances in tabes by Flatow[11] no reference to the occurrence of such changes in the ear; there is also nothing to be found on this point in the otological literature. These changes might be found oftener at the necropsies if they were looked for. Gellé, *e.g.*, in his studies on Ménière's disease in the *Annales des maladies de l'oreille*,[12] describes such a case without calling attention to the connection.

The patient, æt. forty-two, had suffered from tabes since her fourteenth year. Examination: Membrana tympani very translucent, hearing power moderate (watch, right, 6, left, 12 *cm*), tuning-fork heard equally well on either side. Necropsy: Membrana tympani normal; mucous membrane of the tympanic cavity dry, sclerotic, gray. Several synechiæ existed, and besides this the tissue of the fenestra rotunda was thickened, and in fact as hard as cartilage. All the changes were the same in the two ears. Internal ear: anæmia of the lig. spirale, unusual thinness and transparency of the lamina spiralis; atrophy of the nerve elements of the basilar membrane; auditory nerve normal. Perhaps one of the two often quoted cases of Lucae[13] belongs here. The left ear, according to the patient, had been hard of hearing since his childhood; the right ear, however, had become affected since the tabes began. There were sclerotic changes in both ears, with an anchylosis of the stapes. While for the right ear another cause may be assumed, the left showed merely the picture of an autochthonic sclerosis of the lining membrane of the tympanic cavity.

It should be remembered further that, in cerebral affections, here and there sclerotic changes have been observed in the middle ear. Reports of the necropsies of cases of this sort may be found, *e.g.*, in Schwartze's Contributions to the Pathological Anatomy of the Ear.[14]

Although a number of facts would indicate the development of sclerotic changes in the middle ear from a tabetic cause, yet numerous observations must be made before this question can be definitely decided.

Another question is, what reason has one for supposing the affection of the ear in a tabetic patient to have been caused by the tabes? The deciding of this question, in a disease whose commencement is often overlooked, and usually dates further back than the patient supposed, is very difficult. One would, however, be justified in referring the ear trouble, in all probability, to the spinal disease, when the disturbance of hearing is first noticed after the undoubted beginning of the tabes, no aural disease having previously existed, providing that the otological examination corresponds to the clinical history. These postulates hold good for the two cases given above.

I shall not venture to decide in regard to the electric reaction of the auditory nerve, on which Gradenigo lays great stress in his last work, with reference to the early diagnosis of affection of the auditory nerve in central diseases, whether it be of importance. With the exception of the interesting results in the case Wehrfeld, and a subjective sensation of sound aroused once only, perhaps by firmer pressure on the tragus, in another tabetic patient with atrophy of the optic nerves, I have not been able to get a reaction from the method described above, although in some cases the current was increased up to sixteen milliampères. I cannot confirm the statement of Gradenigo that, especially in cerebro-spinal diseases with amblyopia or amaurosis, an excitability of the auditory nerve is found. For, of eight cases of atrophy of the optic nerve found among the twenty tabetic cases—forty per cent. of those examined,—there was only one which showed a reaction with six milliampères. It is also strange that in the case of Lau, where there was a marked hyperæsthesia for musical sounds and for noises, an electric reaction could not be obtained by three milliampères, although cerebral symptoms were already present.

In this paper of Gradenigo, there is a statement of Dr. Sune y Molista, on some paradoxical conditions of hearing in deafness from tabetic or central causes. In these affections in cases where a screaming voice is heard only very near the ear, the sound of the words, without the detail of the articulation, is still perceived, and also the scratching of a pen, the striking together of two metal pieces, etc. The observations are certainly correct; the author is wrong, however, in supposing these paradoxical conditions, as he calls them, to exist only in deafness from central causes. They are often found in deafness from various causes, especially sclerosis. Such patients very often say that they hear the striking of the tuning-fork, but not its tone.

I would remark incidentally that I have not been able to confirm the observation of Van Stein,[10] who found in a hysterical woman, with anæsthetic zones in the head, that the sound of the tuning-fork, when placed on these points, was not heard. In two patients with marked lessening of

pain- and touch-perception on one side, no difference was found in the sound-perception of the two sides. Whether, for the production of this condition, a lessening of all the sensibilities is required, is very questionable.

SUPPLEMENTARY.

While I was still engaged in my investigations, a paper appeared in the last number of the *Arch. f. Psychiatrie* by Dr. Alessandro Marina, On the Symptomatology of Tabes Dorsalis, with Special Reference to the Ears, Larynx, and Pharynx. At the request of Marina, Morfurgo examined the ears of forty tabetic out-patients, and his results showed that only 17.5 % of these had normal hearing, and that 72.5 % were affected with probable disease of the internal ear. Although one must allow that chance has much to do with the composition of the material, since, among Marina's tabetic patients, 82.5 % were hard of hearing, while Rosenthal found only 10 %, Voigt, 2 %, and I 25 % (10 % of these nervous), still one must object to his statement that 72.5 %, or about 90 % of those hard of hearing, had a nervous affection. For the most of Marina's cases the principles laid down above for the diagnosis of nervous deafness do not hold. In all his cases there is wanting a statement as to the results of Rinne's experiment and the duration of the perception of acute tones, where the latter were ever employed in the examination. Only in four cases (xiv., xv., xxix., and xxxvi.) is the statement made that the bone-conduction was markedly decreased, and these cases might, with some probability, be considered nervous. The 72.5 % of cases of nervous deafness may then be reduced to 10 %, which is the result to which I came.

Moos, after looking over this paper, had the kindness to add that Chataigner[16] has recently studied the disturbances of hearing in tabes, and has reached the following conclusions: "The disturbances on the part of the ear may be of varying intensity. They are caused by a hyperæmia of the labyrinth, which may depend on a direct irritation of the auditory nerves which extends from the centre toward the periphery, or of other nerves which exercise a vaso-motor

influence on the vascular system of the ear. The deafness may be, in the former case, total; in the latter, partial. The disturbances of hearing may be the first symptom of the tabes." Herein lies a confirmation of the results of my investigations, since here the auditory disturbances in tabes are referred to vaso-motor influences, which, for their part, must lead to trophic changes.

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A CONTRIBUTION ON THE QUESTION OF THE PART TAKEN BY MICRO-ORGANISMS IN OTITIS MEDIA DIPHTHERITICA.

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Translated by James Pedersen, M.D., New York.

A NUMBER of the nineteenth volume of these Archives contains a work by S. Hirsch, on diphtheritic inflammation of the mucous membrane of the tympanic cavity. In the second of the cases described, the tympanic membrane was intact; the tympanic cavity was lined by a firmly adherent fibrinous mass, and filled by muco-purulent secretion. Microscopic examination of the mucous membrane and its fibrinous covering, stained by the Gram-Weigert method, showed the mucosa to be in a condition of "fibrinous necrotic inflammation," and that the superimposed membrane contained numerous colonies of darkly stained cocci between the blue-colored shreds of fibrin.

In harmony with this result, in a case of scarlatinal diphtheritis of the ear, the fibrinous membrane of the tympanic cavity, as stained by the Gram-Weigert method, was found to be in exactly the same condition as portrayed by Hirsch in his description and plate. As opposed to it, however, the examination of a diphtheritic membrane, taken from one of the air-cells of the same preparation, offered an essentially different picture as regards bacteriology. The subject of the research was the temporal bone of a girl suffering from scarlet fever and scarlatinal diphtheria, who was brought to the Hauner Hospital for Children in the latter part of February, 1888.

We are indebted to the kindness of Professor Ranke for the history, an abstract of which is as follows:

Elizabeth K., seven and one half years old. Diagnosis: Scarlatina maligna and septicæmia. Patient was taken sick on February 10th with fever and pain in throat. Rash appeared and discharge from the nose began February 12th. Since February 14th she has been hard of hearing. On February 19th, discharge from both ears. From the first the throat affection was attended with the formation of an exudation. On February 20th urine contained a trace of albumen. Then on February 24th, large pieces of membrane came away from the nose. There was diffuse swelling of left arm. Pulse very weak. Death occurred on the evening of February 25th.

The post-mortem examination, made in the Pathological Institute, February 26th, thirteen hours after death, which Professor Bollinger kindly placed at our disposal, presented the following anatomical features: "Septicæmia; ulcerative processes in the vault of pharynx; commencing lobular pneumonia of the right lower lobe; moderate hydrothorax and hydropericardium; cloudy swelling of the liver and kidneys; spleen, pancreas, and mesenteric glands enlarged; moderate ascites." From the minuter examinations we quote the following: "The surface of the tonsils had ulcerated; the tonsils themselves were deeply congested, the exudation was slight. The mucous membrane covering the epiglottis and the upper part of the larynx was grayish-red in color, that covering the lower part of the larynx and the trachea was deep-red, but without exudation."

The examination of the left temporal bone was made on February 27th, by Professor Bezold, to whom I am indebted for the following account:

"The anterior half of the pyramid was broken off obliquely; both Eustachian tube and anterior wall of the auditory canal were gone; the cochlea was split in a transverse direction, was not injected, and its scalæ were empty.

"The external auditory canal, which had been broken open in the removal of the bone, had its bony portion filled with diffuse, red, glistening granulations. In the innermost zone and up to the periphery of the tympanic membrane the

posterior wall was bare for a width of 3 to 5 *mm.* In the neighborhood of the tympanic membrane there was an irregular, thick, white, fibrinous mass; it embraced the whole lower half of the seat of the drum and covered the inner surface of the tympanic cavity. The lower end of the handle of the malleus lay embedded in this mass. More superiorly, the hammer and the short process of the malleus were free and surrounded above and in front by healthy red granulation-tissue. Directly adjacent to the handle and extending backward from it to the bare bony posterior wall of the auditory canal, was an opening leading into the tympanic cavity, in which the crus of the anvil lay exposed.

On opening the tympanic cavity from above, the lumen of the tympanum, of the antrum, and of the aditus ad antrum was found partly empty, its walls everywhere covered with creamy pus. When touched with a probe the walls seemed everywhere devoid of mucous membrane. The hammer and the body of the anvil were also bare; the articulation between them was intact, and the long crus of the anvil was in direct relation with the stirrup. The mobility of the latter was normal. The vicinity of the stirrup and that of the fenestra rotunda were covered in by a fibrinous membrane.

The mastoid cells bordering upon the antrum were well developed and exhibited the same condition throughout as the tympanic cavity and the antrum. Their lumen was filled chiefly with pus which, when washed away, left bare walls, pierced here and there by very fine spiculæ of bone. Only in isolated spots whitish patches still clung to the walls, while here and there could be seen a dirty-red granulating mucous membrane.

A vertical section through the middle of the mastoid process opened a cavity the size of a small bean filled with pus. Its walls were bare for the most part. At the level of the spina supra meatum this cavity was placed 8 *mm* behind the former. From it the sound passed through a narrow bony canal directly into the antrum. Above this cavity, directly beneath the outer covering of bone, was a second, about as large as a pea, separated from the first

only by a thin bony partition. This cavity was occupied by a firm, fibrous plug, which, however, was easily removed with a probe. The cavity was then seen to be lined by a moderately thickened, slightly injected mucous membrane. Aside from these two cavities, the mastoid portion of the temporal bone in the plane of section consisted throughout of a spongy substance somewhat greenish in color, but otherwise only slightly changed. The larger cavity was 9 *mm* from the apex of the mastoid process.

A specimen of the fibrinous mass which filled the tympanic cavity was examined in the fresh state. It consisted mainly of pus cells, either free or held together by a network of fibrin. The hyaline threads of the network were coarser than those found in a croupous exudation in the auditory canal (in cases of otitis externa crouposa). The whole specimen was studded with micrococci.

The fresh, moist diphtheritic membrane which, as mentioned above, was easily shelled out from the second cavity, was hardened in alcohol, stained in borax-carmine, imbedded in paraffin, divided into sections, and then put through Weigert's method of staining fibrin. Even when magnified only one hundred times, a beautiful, sharply outlined, fine, blue network of fibrin was seen, showing a distinct alveolar structure and containing some larger spaces. Distributed irregularly and very unevenly over the cut surface were *numerous red-tinged leucocytes, generally with single nuclei and nearly of one size and shape.* But, on the other hand, *no micro-organisms were found*, even by aid of the best immersion lens. Professor Roth (Basel) had the kindness to examine two sections, and he corroborated my result.

This contribution was suggested by Hirsch's work, quoted above, and also by the opinion of O. Heubner as given in his reports on Scarlatinal Diphtheria and its Treatment (*Sammlung klin. Vorträge*, v. Volkmann, No. 322), an opinion which holds that the presence of streptococci in the diphtheritic parts—living tissue as well as fibrinous membrane—is to be explained as the result of a secondary entrance from without (through the air and the free contact of secretions with the same), and therefore that their devel-

opment must be considered a secondary process which has nothing to do with the etiology of the diphtheritic inflammation, but which denotes the beginning of the septic infection.

Our case deals with a diphtheritic membrane which had plainly formed in a cavity that was closed off on all sides, and after the narrow and only outlet had been completely occluded by the inflammation, and where, therefore, a secondary invasion by bacteria, either through the air or through secretions, was hardly possible. Thus we do not hesitate to proclaim our result, seeing that it is a further evidence in favor of the teaching of Heubner. In considering the above conclusion we must, of course, look upon this membrane as a recent one. That it began to form after so long a time, relatively speaking, must be noted as remarkable. It is true that for the ear there is wanting, in this respect, satisfactory observations as to the length of time within which subsequent growths of diphtheritic membrane can take place. In the lungs, Sanne and Peter (" Handbuch der Kinderkrankheiten," Bd. iii., 2te Hälfte, S. 164) found, among 256 cases of diphtheria of the larynx upon which autopsies were performed, only two in which there were croupous deposits in the bronchi after the twelfth day of the sickness.

WORDS AS TESTS OF THE HEARING POWER AND THEIR DIFFERENTIAL DIAGNOSTIC VALUE.

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Translated by Dr. Ward A. Holden, New York.

TWO years ago at the Wiesbaden meeting of naturalists, in the discussion concerning methods of testing the hearing power, many persons concurred with me in believing [1] "that human speech is the most complete but also the most complicated of all the methods of testing the hearing power.[2]"

They were agreed that it was necessary and desirable to choose particular test-words and to recommend these to German aurists for general use. The words suitable for such tests could not then be agreed upon, since it was necessary first to make an acoustico-physiological separation of essential and unessential sounds. In the meantime I have endeavored to make this separation, and I present now my results.

Hitherto we have for the most part tested the ear only as to its quantitative hearing distance—*i.e.*, we find the distance within which a chosen word whispered or spoken aloud can be perceived. I regard it as indispensable for the differential diagnosis that we make also a qualitative test, that the breadth of hearing be found, viz., what range of tones, acute, medium-pitched, or grave, are heard by the patient well, with difficulty, or not at all, as compared with the normal perception.

[1] *Cf.* "Sprache und Ohr," Braunschweig, 1871.
[2] *Cf.* Journal of the 60th Meeting of German Naturalists, p. 330.

With the aid of the consonants *R* lingual, *B*, *K*, *T*, *F*, *Sch*, *S*, and *G* soft, which I have called self-sounding, we can discover fine differences in perception when we take the acoustic value of the sound rightly. I would not have the tests with the tuning-fork neglected; but we must admit that this takes more time and trouble, and gives more intense sounds to the diseased ear than the consonants, and besides seems to the non-musical ear more incomprehensible and inaccurate than the spoken words.

The misunderstanding of speech by the hard-of-hearing for the most part depends on the faulty perception of the self-sounding consonants, while the vowels, on account of their more considerable strength of tone and the greater amplitude of their vibrations, are still perceived by the most diseased ears. The tone-borrowing consonants, as I have termed them, *L*, *M*, *N*, and *H*, need not be taken into consideration, since they do not possess a peculiar tone acoustically definable, but merely modify the beginning and the end-tones, the timbre and the resonance of the vowel, and are therefore in linguistics called "resonants." By this exclusion the number of test-sounds is materially lessened. We need select for the tests only a few words, those in which the self-sounding consonants predominate—*e.g.*, as in the word "Messer" the *S*-sound.

The normal human ear is most sensitive to the most acute tones of speech, so that those tones which lie in the middle portion of the iv. octave and approach the proper tone of the auditory canal and membrana tympani (e^{iv} to g^{iv}) may caues under certain circumstances the sensation of pain.[1] The faintest pianissimo of the violin up to c^{vi} is distinctly heard in a large hall to the last seats. I believe that the fibres of the zona pectinata, which vibrate in unison with the most acute tones, are the smallest and most delicate.[2]

This extraordinarily fine adaptation of the human cochlea for acute and the most acute tones presupposes the exact

[1] So a light hissing mingled with the applause of a large audience is often heard by the performers with unpleasant distinctness.

[2] Schwalbe is also of this opinion when he says in his text-book on the anatomy of the ear: "The inner and outer rod cells seem to be end organs of the auditory nerves, *auditory cells*, which are most probably excited by the fibres of the zona having various lengths adapted to the various tones."

working of the conducting apparatus. The membrana tympani and the membranes of the fenestræ must be normal, in order to be brought to vibrate in unison with the acute and the most acute tones.[1]

Thickening of the lining membrane of the tympanic cavity, adhesions which impede the vibrations of the membrana tympani and the ossicles, and, finally, increased tension of the membrane covering the fenestra ovalis will, in the majority of cases, prevent the perception of the higher tones, and especially the *S*-sound, whose pitch corresponds to from $c^{iv} - c^{v} = 5{,}400 - 10{,}800$ simple vibrations, and is a simple tone with few inharmonic overtones. The lessened perception for the most acute tones is observed in elderly persons, whose sound-conducting apparatus has become somewhat rigid. Physical experiments confirm these observations. Ten years ago I experimented with the first Edison phonograph which was sent to Europe, and found that with the strong vowel *A*, rich in harmonics and having a fairly grave fundamental tone (b^{ii}) the needle drew the deepest and largest figures on the cylinder, since the membrane then had the most ample vibrations, while with the *S*-sound numerous small almost punctiform shallow impressions were made.[2]

The latest phonograph has improved somewhat as to the timbre of the sounds, yet it reproduces most defectively the *S*-sound. This is the case in still higher degree with the telephone, whose plate is more rigid than an animal membrane, and therefore reacts with difficulty to the acute *S*-sound.

The belief that the smallest fibres of the zona pectinata, which vibrate in unison with acute tones, lie in the first turn of the cochlea, and that the larger fibres adapted to grave tones lie near the cupola, is indicated, among other facts, by the observation of Moos and Steinbruegge[3] who

[1] V. Hensen says touching this point ("Textbook of Physiology" by Hermann, vol. iii., 2, p. 112): "The limit upward does not seem to be reached with 40,000 (simple) vibrations. The most acute tones can scarcely be made strong enough to cause the human membrana tympani, which is as I believe somewhat rigid, to vibrate in unison."

[2] *Cf.* the figures in my paper: Experiments with the Edison Phonograph, these ARCHIVES, vol. viii., p. 58 *et seq.*

[3] On Nerve Atrophy in the First Turn of the Cochlea; read at the 2d Internat. Otol. Congress, Milan, 1880.

found atrophy of the nerve in the first turn of the cochlea, with changes in the tympanic cavity, in a case in which the patient in life had not been able to hear the more acute tones of the scale, but had retained the perception for the grave and gravest tones.

The *Sch*-sound is relatively better perceived than the *S*-sound, since it has two grave harmonics, and therefore has a fuller sound; it represents the pleasing harmonic chord ($f^{\sharp\,iv} + d^{iv} + a^{iii}$); *S* and *Ch* soft are united in it. Some patients, who, on account of the rigidity and tendinous thickening of the conducting apparatus, are unable to hear the *S*-, perceive of the *Sch*-sound only the *Ch* soft, and understand, *e.g.*, instead of the word "Wäsche," "Waege." The *F*-sound, although standing nearest the hissing sounds in pitch (a^{ii}—a^{iii}), on account of its less intensity, affects very slightly the normal ear, and is, consequently, heard with difficulty in various ear diseases. Its inaudibility may, however, indicate the isolated affection of certain cochlear fibres, *e.g.*, hemorrhage, when it alone is not heard near the ear while other sounds of less intensity are perceived. I have reported several cases[1] in which the patients heard all sounds well, excepting the *F*-sound, once after a shot fired in a closed room, another time after a kiss directly on the ear, which was followed probably by a small hemorrhage into the labyrinth. This patient complained of hearing a sound continuously, corresponding in pitch to the *F*-sound.

It is most convenient to divide the test-sounds into three groups: (1) the acute and intense *S*, *Sch*, and *G* soft (this latter sound does not exist in English, but is nearest to our *Sh*), acute *F*-sound of low intensity; (2) the group of the explosive sounds, *B*, *K*, *T*, of medium pitch and intensity; (3) the group of grave sounds of low intensity: the rolling *R* and the whispered *U*. The *B*-, *K*-, *T*-sounds of the second group are termed explosives, since in their articulation the air leaves the mouth with an explosion. They cause, therefore, a forcible inward movement of the membrana tympani, especially in its central portion, as Blake

[1] New Investigations on Tests of the Hearing Power and on Disturbances of Hearing. *Cf.* these ARCHIVES, vol. iv., p. 67.

found in a very interesting series of experiments.[1] They are heard also relatively well by the deaf. In diseases of the labyrinth, according to Dennert,[2] tones of medium pitch are lost far oftener than acute or grave tones. Words like Kette, Teppich, Tante, Kappe, seldom give rise to confusion of the consonants.

The rolling *R*, of the third group, is important as a grave test-sound. Its fundamental tone has only perhaps sixteen double vibrations to the second, and corresponds to C^{-3}. In order to make these vibrations audible, it is best to give the lingual *R*, placing the tongue and lips as for sounding *B-r-r-r*.

For the precise conduction of grave tones with great amplitude of vibration, a pantographic working of the conducting apparatus seems necessary in order that the sound waves shall reach the fenestra ovalis diminished. If the membrana tympani be wanting, this effect is not gotten, and of the *R*-sound only those higher breathing sounds are perceived, which, besides the grave vibrations, are produced by the breath. Patients with defect of the membrana tympani hear therefore frequently *F*, instead of the rolling *R*, *e.g.*, "Fuder" instead of Bruder, rather guessing the final *R*.[3]

Since the defect of the membrana tympani is easily discovered by inspection, the differential diagnostic value of the *R*-sound is slight in defects of the conducting apparatus; the inaudibility of this sound in patients with *intact conducting apparatus*, however, furnishes us important conclusions. When such a person perceives these grave tones relatively poorly, we may conclude that there is disease of the labyrinth, provided that other considerations, especially those regarding the etiology of the disease, point to this diagnosis. I found perception of the gravest lingual *R*- and whispered *U*-sounds wanting and defective after typhus and

[1] Clarence J. Blake (Boston): The Use of the Membrana Tympani as a Logograph and Phonautograph. These ARCHIVES, vol. vii., p. 458.

[2] Dennert (Berlin): On the Analysis of the Organ of Hearing by the Significance of Tones for it. *Berliner klin. Wochenschr.*, 1881, No. 18.

[3] Patients with defect of the membrana tympani perceive the *S*-sound on the contrary very readily, since the smallest vibrations of this sound can cause the membrane of the fenestra to vibrate in unison with them.

other exhausting diseases, as well as after rapidly following parturitions, prolonged lactation, in chronic parametritis, in anæmia and chlorosis, and in affections of the sympathetic system. Disturbances in the circulation of the blood and the consequent defective nutrition of the labyrinth seem to play here an important rôle. Attacks of giddiness and grave subjective sounds (Ménière's symptoms) are found especially in the more severe forms. The relatively unfavorable blood supply which the little internal auditory artery furnished to the labyrinth, makes easy the development of disturbances of nutrition in particular portions of the labyrinth.

In conclusion, I recommend a number of practically useful test-words for the three groups of spoken sounds:

First group.—*(a) Acute and far-sounding hissing sounds, S, Sch, G soft.*

Intensity for the normal ear: *Sch*-sound . . 200 paces.
" " " " " *S*-sound . . . 175 "
" " " " " *C* or *Ch* soft . 130 "

Test words: Messer, Strasse, Säge.

In disturbances of the conducting apparatus, the patient for the most part understands Meter for Messer, Braten for Strasse, Fäden for Säge.

(b) Acute sounds of low intensity, *F*, and *V*.

Intensity of *F*-sound . . . . . 67 paces.

Test words: Feder, Frankfurt.

The patient often understands Leder for Feder, Bratwort or Antwort for Frankfurt. The word " Frankfurt " is especially difficult to understand, because of the combination of consonants.

Second group.—Explosive sounds of medium pitch, *B* (pitch e^{i}), *K* (pitch d^{ii}), *T* (pitch $f^{\sharp\,ii}$).

Intensity: *K*- and *T*-sounds . . . 63 paces.
B-sound . . . . . 41 "

Test words: Teppich, Tante, Kette, Kappe.

Third group.—Grave sounds: *M* (pitch f^{o}), *R* lingual, without voice tone (pitch of the dominant tone, $C^{-3} = 16$ double vibrations to the second).

Intensity: *U* (whispered) . . . . 50 paces.
R (rolled) . . . . . 41 "

The *M*, like the other vowels, is of differential diagnostic value only when whispered.

Test words: Ruhe, Bruder, Ruhrort, Reiter.

Fuder is mostly understood instead of Bruder; the word Ruhrort (whispered) is perceived with great difficulty when disease of the conducting apparatus exists. I have given several test-words for each group, in order that in repeated tests the patient may not so easily guess what he has not clearly heard. It is to be recommended on this account that other than the characteristic words, *e.g.*, numbers, be interposed in the tests.

REPORT OF THE 9TH MEETING OF SOUTH-GERMAN AND SWISS AURISTS HELD AT FREIBURG (BADEN), EASTER, 1889.

OFFICIAL REPORT BY DR. E. BLOCH, FREIBURG (BADEN).

Translated by Dr. WARD A. HOLDEN, New York.

Those present were: BEZOLD, Munich; BISCHOFF, Basle; BLOCH, Freiburg; HEDINGER, Stuttgart; KILLIAN, Freiburg; Moos, Heidelberg; POLITZER, Vienna; SCHUBERT, Nuremberg; SIEBENMANN, Basle; THIRY, Freiburg; WERTHEIMER, Freiburg; WOLF, Frankfort-on-the-Main.

1ST SESSION, EASTER SUNDAY, APRIL 21, 1889, AT THIRY'S HOUSE.

Thiry, after greeting the guests in a short speech, was chosen chairman by acclamation, and Bloch was chosen secretary in the same manner.

It was decided that the next meeting should be held at Nuremberg at Whitsuntide, 1890.

1. Moos, Heidelberg, demonstrated preparations of the petrous portion of the temporal bone of children who had died of diphtheria.

2. The same: *On neuritis diphtherica.*

The affections of the nerves hitherto recognized in diphtheria are of two varieties, hemorrhages and true neuritis. Buhl first described hemorrhages in the intervertebral ganglia and in the roots of the spinal nerves. Mendel saw capillary hemorrhages in the pons and medulla, and W. Krause found a considerable hemorrhage in the trigon. interpedunculare of a child of eleven years dead of diphtheria, where symptoms of naso-pharyngeal paralysis

existed in life (*Neurol. Centralbl.*, 1888, No. 17). A neuritis of the phrenic nerve, in a case of diphtheritic paralysis, was described by Paul Meyer, who found the medullary substance split up, the nuclei of the sheath of Schwann increased in number, the nerve-fibres totally destroyed and transformed into granular fatty cells.

Both these changes occur in the labyrinth in diphtheritic patients. Moos demonstrated a number of preparations showing defects in the trunk of the auditory nerve and in its cochlear and vestibular branches, in which traces of blood could still be seen with the naked eye or a glass. These defects, as the speaker showed in several preparations, may develop without hemorrhage, in consequence of neuritis, and attain a diameter of 3 *mm.*

The neuritis is caused by the migration into the nerve-sheaths of cocci, which in longitudinal sections appear as long stretched-out chains; in cross-sections as strings of pearls. The cocci also enter the nuclei of the sheath of Schwann, and at the same time there is found an increase in the number of nuclei and a hyperplasia of the interstitial connective tissue. The speaker explained in this manner the cases reported of diphtheritic polyneuritis, the mycotic nature of which, though suspected, could not be proved.

3. The same: *On Bezold's disease of the mastoid process, and thrombosis of the lateral sinus.* The paper is printed in the preceding number of these ARCHIVES.

Discussion. BEZOLD in such cases now opens the antrum instead of merely boring through the mastoid process as he formerly advised.

THIRY had seen four such cases. To cure them it is only necessary to remove the cortex of the mastoid process and thoroughly open and drain the pus cavity in the neck. In one case the suppuration extended to the clavicle and to the spinal column, laying bare two lateral processes, yet a cure followed without fever after the operation. One case, in which the disease had lasted four weeks, had been operated on only two days before, an abscess being found under the temporal muscle above the zygoma.

HEDINGER stated that Burkhardt, Stuttgart, had operated seven times; all the cases were cured. In one case the lower half of the mastoid process was sclerosed.

POLITZER believes that the operative method selected should depend upon the objective symptoms in each case. In general, the laying open of the mastoid antrum and the washing through the middle ear has the desired effect, but in many cases the operation

is not successful, since the suppuration continues in terminal cells shut off from the antrum by the inflammatory process, and this leads to a circumscribed periostitis and caries of the mastoid process. At autopsies, POLITZER had repeatedly found collections of pus localized in single mastoid cells or in groups of cells. He spoke of one case observed in his clinic in which, during the course of an acute suppuration of the middle ear and following it, an inflammation with redness and swelling developed in the posterior part of the mastoid process, which could not be checked by energetic antiphlogistic treatment. Through an incision made 3 *cm* behind the concha, the posterior terminal mastoid cells, already broken through, were reached; yet these, as repeated syringing showed, had no connection with the cavity of the middle ear. Repeated scraping and bandaging with iodoform cured the disease. When the pus breaks through at the incisura mastoid, POLITZER cures the cases by opening the antrum, but he has been compelled in a number of cases, in which the symptoms of pus accumulation below the mastoid did not cease on laying open the antrum, to make a deep incision below the mastoid process and free the pus by pressing the scalpel-handle deep into the wound.

BEZOLD finds the morbid process in different cases at different points and in different stages, and even different stages (mucous, purulent secretion), in a single case.

WOLF believes that one should go in whenever pus is known or is supposed to exist.

POLITZER, on the contrary, treats acute cases in the beginning antiphlogistically. THIRY also employs antiphlogistic treatment at first, and later, if necessary, the knife, etc.

4. MOOS. *Demonstration of preparations of colloid degeneration of the m. tensor tympani.*

This may be found in diphtheria, for example, as early as the second day.

5. POLITZER, Vienna. *Demonstration of normal and pathological ear preparations.*

POLITZER demonstrated first several normal preparations in alcohol, in which the entire auditory apparatus—auricle, auditory canal, tympanic membrane, tympanum, Eustachian tubes, semicircular canals, and cochlea could be seen in their natural topographical relations.

POLITZER spoke further of the development of connective-tissue and membranous adhesions in the middle ear, showing a number

of instructive pathological preparations. According to his observations, excessive development of connective tissue may occur in the course of suppurative as well as of non-suppurative middle-ear diseases, but it is found most frequently in the upper portion of the cavity of the tympanum (atticus), which may be filled with a connective-tissue mass enveloping the malleus and incus and extending into the antrum, while the middle and lower portions of the cavity remain free. The tendon of the tensor tympani forms as a rule the boundary between the middle free cavity of the tympanum and the atticus filled with connective tissue. The speaker concludes from this fact that the atticus differs from the middle and lower cavities in certain peculiarities which perhaps reveal themselves in man only in pathological conditions ; and he remarks that in many animals there is a separation, though incomplete, between the atticus and the remainder of the cavity.

POLITZER showed, in a second series of preparations, the complete filling up of the cavity of the tympanum, after chronic suppuration of the middle ear, by connective-tissue masses, with cicatricial adhesions between the remaining portion of the membrana tympani and the inner wall of the tympanum, in which manner a pocket is often formed extending from the margin of the perforation into the tympanum toward the promontory.

POLITZER exhibited also a preparation of the right ear, in which, in the posterior bony wall of the external canal, an opening 4 *mm* high by 3 *mm* broad led into a passage narrowed in its middle part, which, running through the upper portion of the mastoid process, opened again on the external surface, just behind the ear, by a perforation 6 *mm* high by 4 *mm* broad. The canal and its openings were covered with skin. This case then showed a fistula, formed by a previous cario-necrotic process in the bone, which opened into the external canal and on the outer surface of the mastoid process. In a similar case observed by POLITZER, the canal was filled with a cerumen-like mass, which, being removed, the probe could be freely passed through the fistula into the auditory canal.

In conclusion, POLITZER demonstrated microscopic preparations of a case of persistent perforation of the membrana tympani after suppuration of the middle ear. Frontal sections showed the stratified epithelium, horny in its upper layers, of the external canal and the outer surface of the membrana tympani. At the margins of the perforation this epithelium had proliferated and

continued over the mucous membrane of the tympanum. POLITZER believes that this proliferation of the epithelium over the margins of the perforation must frequently be the cause of the persistence of the perforation.

6. BEZOLD, Munich: *On cholesteatoma of the middle ear.*

The paper is printed in this number.

Discussion. MOOS thanked the speaker, and concurred in the views expressed.

POLITZER said that Bezold's theory of the development of cholesteatoma, by the extension of the epidermis of the external canal into the tympanum and the mastoid process, was supported, as far as many of the cases are concerned, by preparations in his collection. Such an encroachment of the epidermis of the canal and outer surface of the membrana tympani upon the tympanum, could clearly be seen in POLITZER'S preparation, showing epithelial proliferation at the margins of a persistent perforation. POLITZER believes, however, that, from the pathologically changed mucous membrane of the middle ear, an epithelial proliferation may occur, leading to the formation of the laminated tumor called cholesteatoma.

This view is supported by a preparation in his collection which shows a smooth, pearly tumor, as large as a bean, in the upper portion of the tympanic cavity, which doubtless developed from a cyst of the mucous membrane. That such masses may develop from chronically inflamed mucous membranes is proved by the reports in the literature of cholesteatoma of the frontal sinus, in which cases the development proceeded quite independently of the outer epidermis.

As regards the frequent occurrence of perforation of Schrapnell's membrane in conjunction with chronic suppuration in the mastoid antrum, POLITZER referred to the statements made in his last work, "The Anatomy and Histology of the Human Organ of Hearing," Stuttgart, 1889, that, at times, in normal preparations, there is a membranous network stretched in the mastoid antrum, adherent to the bony walls, extending uninterruptedly into the tympanum and connected with the bands and mucous-membrane folds of the malleus and incus.

According to POLITZER, there can be no doubt but that inflammatory processes in the malleo-incus niche may extend, through the medium of this network, into the antrum, and *vice versa*, from the antrum into this niche, as many preparations in his collection would indicate.

2D SESSION: EASTER MONDAY, APRIL 22, 1889.

7. Moos, Heidelberg, demonstrated preparations of tertiary syphilis of the middle ear in the bony portion of the Eustachian tube, and in the promontorium.

8. SCHUBERT, Nuremberg, reported a new case of the growth of mould in the nose. The patient complained of an offensive nasal discharge. The mucous membrance of the nose was covered on both sides with a thick gray-green pulpy mass, emitting a peculiar odor unlike that of ozaena. Under the microscope this mass proved to be composed of a richly branched mould mycelium, and of numerous cylindrical conidia on the ends of the mycelium twigs, without an especially differentiated fruit organ. The conidia were from three to four times as long as broad, curved slightly for the most part, without nuclei, and rounded at the ends. Prof. FERDINAND COHN, to whom specimens were sent, said that an accurate classification of the mould would be impossible unless it be cultivated, for which purpose the preparations were unfit, but that a mould with cylindrical conidia had not hitherto been observed in vertebrate animals, and that this case was therefore unique. Such forms are common in insects; for example, the botrytis bassiana, the mould of the muscardine disease of silk worms, has the same appearance as this nasal mould. The speaker gave a short account of the development of botrytis bassiana, according to De Bary, showing his illustrations in the *Botanische Zeitung*, 1857. Excepting a review in the *Archiv. f. klin. Med.*, vol. xxxvi., p. 162, of the finding of an aspergillus fumigatus, there seems to be no further report in the literature of the development of higher moulds in the nose.

9. SIEBENMANN, Basle, exhibited a corrosion preparation of the macerated frontal bone of an infant, made of Wood's metal, after his method described in the "Transactions of the Natural History Society of Basle," 1889. Beside the bony aquæduct and the canalis Cotunni, this preparation shows clearly the accessory vessel canals of both aquæducts, then pictured and described for the first time. This corrosion proceeding furnishes as favorable results as could be expected of a method so inexpensive.

10. KILLIAN, Freiburg, made a short preliminary report of his comparative anatomical and embryological investigations on the vertebrates, for the purpose of determining the morphological significance of the ear muscles in man, and communicated his results.

11. POLITZER, Vienna: ***Demonstration of low-pitched tuning-forks.***

The speaker presented a number of tuning-forks, made after his plans by Blumenkranz, in Vienna, which are cheaper and lighter than those previously made. The quadrilateral prismatic forks are 32 *cm* long, 1½ *cm* broad, and 7 *mm* thick. POLITZER had made also cylindrical rod-shaped forks 30 *cm* long and 12 *mm* thick, which vibrate from 1½ to 2 minutes. Neither the cylindrical nor the prismatic forks give audible overtones, although they have no clamps on the prongs. This was secured, according to POLITZER, by having the lower portion of the fork filed thinner, and one entire prong made somewhat more slender than the other.

THIRY, Freiburg: ***On the decrement of vibration in tuning-forks.***

The determination of the power of hearing by means of the test of the vibrating forks, can be considered accurate only when the nature of the vibration is known and taken into account. According to physical theories, the forks, while vibrating, must diminish in their amplitude of oscillation in a geometrical progression. Hensen ("Physiology of Hearing"), after experimenting with three forks, arrived at the conclusions that the logarithmic decrement of the amplitude of oscillation is not a constant, and that the amplitude decreases irregularly. The experimental proof was not given.

Jacobsen (*Archiv. f. Physiologie*, 1887, p. 476) was the next to study vibrating forks. He arranged a tuning-fork so that it recorded its vibrations on a sooted cylinder, and from the microscopic measurement of the wave elevations in various portions of the curve, he computed the proportionate degree of the successive amplitudes and their logarithmic decrement, and came to the conclusion that with large amplitudes the decrement varies from a constant more than can be accounted for on the ground of unavoidable errors in the observations, and that it becomes a constant only in the case of small amplitudes; this variation being so slight, however, that in determining the power of hearing it need not be taken into account.

The results that Jacobson obtained later by photography are unknown to the author.

Barth [1] published his investigations in the same year that Jacobson's were published. Barth fixed a globule of mercury to one prong of his fork, and this being set vibrating, he photographed from second to second the microscopically magnified oscillating

[1] These ARCHIVES, Vol. xvii., No. 2.

image of the illuminated globule. From the length of the line photographed he computed the decrement. He found that the decrement of vibration was under similar conditions always a constant.

These contradictory results made a new study of the question desirable, since every aurist would wish to be as conversant as possible with the properties of his instruments, even though such knowledge did not, as in this case, increase the practical usefulness of the instruments. I requested the assistance of Prof. Koch, and the following investigations were made jointly by us in the physical laboratory at Freiburg.

The first experiment was with the large *c*-fork of Lissajous; this was placed on a stand, and by the placing on it of heavy weights, an attempt, not altogether successful, was made to render it immobile. On the mirror was projected a point of light, later a line of light, produced in part by an Argand burner, in part by a lime light, and the image was compared with a transparent vertical scale. On drawing a violin bow across the fork, the point of light elongated itself into a line of light which gradually grew shorter; the time was then measured which elapsed before the lower end of the line of light, previously placed at zero, passed through one 2 *mm* division of the scale. We give the results of this series of experiments in the following short and comprehensive form: If the vibrations of the fork decrease after the manner of a geometric series, the same length of time should elapse before the amplitude of the oscillations becomes lessened to one half, as must elapse before from one half it becomes lessened to one fourth, an equal time to one eighth, etc.

The experiments gave this result: In a series of experiments with the point of light the regression from the eighth division to the fourth required 13.25 seconds; from the fourth to the second, 14.33 seconds; from the second to the first, 14.56 seconds (the average of seven experiments). Another series of experiments with the line of light gave as the corresponding numbers 11.65, 15.86, 16 seconds (six experiments). The other five series, each of which included from six to twelve experiments, gave similar results.

These figures show that a shorter time is required for the elongation to decrease to one half than for it to decrease from one half to one fourth, and similarly from one fourth to one eighth; the decrease of oscillation with greater amplitudes is somewhat

more rapid than would correspond to a geometric series ; with smaller amplitudes the periods of time are so nearly equal that the variations may be ascribed to unavoidable errors in observation. Small amplitudes decrease then according to the law of the geometric series. The results of Thiry's extensive investigations are as follows : *The logarithmic decrement of a vibrating tuning-fork is not a constant, but the decrease in vibration with large amplitudes is more rapid than the geometric series would require ; in small amplitudes it becomes approximately a constant, and the amplitude at which this occurs differs with different forks.*

To determine exactly the power of hearing with the tuning-fork, it is necessary first to have always an equal amplitude of vibration in the beginning, and second to know the manner of decrease in vibration for the fork in question.

If in place of determining for every fork the manner of its decrease in vibration, we assume that this follows the law of the geometric series, the errors are so slight, as Jacobson showed, that they may be practically disregarded. The variation from a standard amplitude of vibration in the beginning is not of the importance that it might at first glance seem. It is doubtful whether such errors need be taken into account in practice, when we remember with how little accuracy it is ever possible to make tests of the hearing power.

14. The same : *Suppuration of the ear simulating gangrene of the lung.*

I was recently invited to be present at the autopsy of a girl æt. ten, who since her second year had suffered with suppuration of the ears following measles, which was slight on the right side, the secretion on the left, however, being abundant and fetid. I had been consulted years ago for this trouble, but the treatment advised had been employed only a short time. In the child's tenth year a severe pain came on in the left side of the neck, and after some days high fever followed with chills and persistent headache. A little later there began a distressing cough lasting day and night, with the expectoration of an offensive discolored sputum, and shortly after there was shortness of breath, and râles were heard in the left lung, and a diagnosis of gangrene of the lung was made. The autopsy showed fibrinous pleuritis of the left side, and bronchitis and hemorrhagic infarcts on both. The sputum came from the ear. The carious excavated mastoid process opened outward by two circular perforations 7 *mm* in diameter,

and opened also into the sulcus by a perforation equally large. About the outer perforation and from it on the base of the skull toward the foramen magnum, the bone was fenestrated and covered with a thin layer of pus. From the posterior margin of the mastoid process a broad fistula passed behind the sterno-cleido-mastoid muscle nearly to the clavicle, opening in its passage, by a perforation 4 *mm* in diameter, into the left side of the larynx. The fistula and the cavity in the mastoid process were filled with a pus similar to that expectorated. The sinus walls in the fossa were markedly thickened, and the lumen of the sinus was narrowed but showed no signs of thrombus. No further pathological condition was found within the cranium or in any other part of the body.

REPORT ON THE PROGRESS OF OTOLOGY DURING THE SECOND HALF OF THE YEAR 1889.

ANATOMY, PATHOLOGY, AND PHYSIOLOGY OF THE EAR AND NASO-PHARYNX.

By AD. BARTH, Berlin.

Translated by Dr. Ward A. Holden, New York.

I.—ANATOMY.

a.—EAR.

1. Sarasin, P. On the organ of hearing in the blind worm of Ceylon, Ichthyaphis glutinosa. *Sitzungsber. d. Ges. naturf. Freunde zu Berlin*, 1889, p. 137.

2. Hennicke, C. R. The organ of hearing in birds. *Monatsschr. des Deutsch. Vereins zum Schutze der Vogelwelt.*

3 His, Wilhelm. On the anatomy of the lobe of the ear. With one plate. *Arch. f. Anat. u. Physiol.*, anat. Abth., 1889, p. 301.

4. Ranke, H. A case of malformation of the ear. *Münchener med. Wochenschr.*, 1889, No. 35.

5. Morgan, J. H. Supernumerary auricles. *Illustr. Medical News*, London, 1889, p. 265.

6. Israel, O. Congenital cleft of the lobe of the ear. *Arch. f. path. Anat. u. Physiol.*, vol. clxx., p. 2.

7. Gray, Robert (student of anatomy, Edinburgh). The external auricular opening and the external auditory meatus in the Greenland right whale, balæna mysticetus. *Jour. of Anat. and Physiol.*, 1889, vol. xxiii., p. 300.

8. SCHWALBE, G., Strassburg. To what extent is the human auricle a rudimentary organ? With one plate. *Arch. f. Anat. u. Physiol.*, anat. Abth., Supplement Band, 1889, p. 241.

9. GRADENIGO, J. The auricle viewed from an anthropological standpoint. (Mém. lu au congrès intern. d'otol., etc., de Paris, Sept., 1889.) *Ann. des mal. de l'oreille*, etc., vol. xv., p. 536.

10. —— On the morphological significance of the ossicles of the ear in reptiles. *Zoölog. Anzeiger*, 1889, vol. xii., p. 336.

11. HAHNE, A. (Inaug. Dissert., Göttingen). On fistula auris congenita. Printed by Vandenhoeck and Ruprecht, Göttingen.

12. LEVY, E., and SCHRADER, M. E. G. (from the policlinic for diseases of the ear and the laboratory of the medical clinic, Strassburg). Bacteriological studies in otitis media. *Arch. f. experiment. Path. u. Pharmakol.*, vol. xxvi., p. 223.

13. GRADENIGO, J. A contribution to the bacteriology of purulent inflammation of the middle ear. (Mém. lu au congrès intern. d'otol., etc., de Paris, Sept., 1889.) *Ann. des mal. de l'oreille*, etc., vol. xv., p. 526.

14. RÜDINGER. On the development of the membranous semicircular canals. With one plate. *Sitzungsber. d. k. b. Akad. d. Wissensch. zu München*, 1889, vol. 1888, p. 493.

15. HIS, W. On the development of the acustico-facialis region in man. With two plates. *Arch. f. Anat. u. Physiol.*, anat. Abth., Suppl. Band, 1889, p. 1.

16. HABERMANN, J. On nerve atrophy in the internal ear. (From Prof. Chiari's path. institute at the German university at Prague.) *Zeitschr. f. Heilk.*, vol. x., p. 368.

17. GRADENIGO, J. The anatomical lesions of the auditory organ in intracranial affections in general, and in the various forms of meningitis. (Mém. lu au congrès intern. d'otol., etc., de Paris, Sept., 1889.) *Ann. des mal. de l'oreille*, etc., vol. xv., p. 528.

18. SCHULTZE, Fr. Total deafness and meningitis. *Virchow's Arch.*, vol. cxix., p. 1, 1890.

19. BAGINSKY, B., Berlin. On the origin and central course of the nervus acusticus in the rabbit and the cat. With one plate. *Virchow's Arch.*, vol. cxix., p. 87, 1890.

20. BAGINSKY, B. The course of the posterior root of the nervus acusticus and the relations of the striæ medullares. *Vortr. in der Berliner Ges. f. Psych. u. Nervenkrankh.*, Nov. 11, 1889.

21. VON MONAKOW, Zurich. Striæ acusticæ and lower lemniscus. *Tagebl. der 62. Vers. deutscher Naturf. u. Aerzte in Heidelberg*, 1889, p. 511.

22. MUNK, H. On the central organs of sight and hearing in the vertebrates. *Sitzungsber. der Kgl. preuss. Akad. der Wissensch.*, 1889, vol. xxxi.

3. HIS speaks of the auricle especially in its comparative anatomical relations, and divides it for this purpose into an upper, hinder, and lower ear. The latter includes all the parts lying underneath the concha. He states that even this hitherto much neglected portion of the auricle has its peculiar form. It also has a cartilaginous skeleton, the lingula auriculæ, which forms a direct continuation of the cartilage plates of the hinder ear.

7. This short paper deals with the form and the closing apparatus of the auricle of the balæna mysticetus.

8. Physiologists hold the human auricle to be a more or less rudimentary organ of little functional service. SCHWALBE wished to determine, from an anatomical standpoint, what part of the auricle is to be considered as reduced. If in the ear of a six-months' human embryo a line be drawn from the upper insertion through the posterior end of the anti-tragus to the posterior inferior margin of the ear, the auricle will be divided into two parts: the bossy portion and the free aural fold. The latter only, in comparison with the ear of the ass and the cow, is markedly reduced. In shy animals it develops into a long, spoon-like sound-receiver, while under other conditions of life, as, for example, in the animals which live in the water, or under the earth, it shrinks to a small curved fold. Adherent ears are to be considered as more reduced than free ears. The cartilage of the auditory canal in man, which is originally composed of three pieces, may be considered as degenerated into a whole without motion of its parts. The spina helicis is a true rudimentary organ. The fact of the muscles of the human ear being more or less rudimentary is treated as thoroughly as are the other points in question. In conclusion the author asserts that, after all, the human auricle is not altogether reduced in comparison with that of the ass and other mammals, but that there is even a development of form by the adding of the lobe. Since the auricle is but partially reduced, its finely modelled form would indicate a physiological function. The problem of its development is yet to be solved.

9. GRADENIGO has observed the form of the auricle in about 15,000 men and 10,000 women, and finds the following relation:

	Men, Per cent.	Women, Per cent.
Adherent lobes	28	22
Free lobes (oreilles à anse)	12.15	6
Wildemuth's form	6	9.12

Disregarding sex, the form varies also according to locality, station in life, and age. The free lobe is found more frequently in children (25 per cent.) than in adults (12.15 per cent.).

12. After a short review of the literature the authors describe their own investigations. These were on 14 cases of acute otitis media, among them 10 paracenteses and 2 chisellings; further, 2 cases of suppuration after cholesteatoma of the middle ear, one of which resulted fatally, by causing a purulent meningitis. In the 10 paracentesis cases there were found as pure cultures: the diplococcus pneumoniæ three times, the staphylococcus pyogenes albus three times, and the streptococcus pyogenes once. As a mixed infection: the diplococcus pneumoniæ with the streptococcus pyogenes once, the diplococcus pneumoniæ with the staphylococcus pyogenes albus once, and the staphylococcus pyogenes albus with the staphylococcus cereus albus once. Of the 4 cases in which perforation had occurred spontaneously, 2 fresh uncomplicated cases showed: the streptococcus pyogenes once, and the streptococcus pyogenes with the staphylococcus pyogenes albus once. In the 2 other cases the mastoid process was trephined; one of these showed the streptococcus pyogenes and one the staphylococcus pyogenes albus with the micrococcus tetragenus. In the case which resulted fatally a diplococcus was found, similar to that of pneumonia in appearance, but different from it in its cultures, which developed at the ordinary temperature of the room.

The authors come to the conclusion that no light is thrown on the probable course of an aural disease by finding certain microorganisms. A given coccus may cause a slight local irritation at one time, and a severe general infection at another. In three later cases of inflammation of the middle ear, which rendered trephining of the mastoid process necessary, the pus gave a pure culture of the staphylococcus cereus albus.

13. GRADENIGO in 4 fresh cases of inflammation of the middle ear found the pneumonia coccus of Fraenkel 3 times, once mixed with the staphylococcus pyogenes aureus and albus; the staphylococcus pyogenes aureus and albus were found alone once. In 4 cases of old fetid suppuration of the middle ear he found the

proteus vulgaris of Hauser 3 times, once with the staphylococcus pyogenes aureus and albus; the staphylococcus pyogenes aureus and albus were found alone once.

14. The semicircular canals do not develop, as was formerly supposed, from a fold-shaped pocket in the aural vesicle and a consequent partial adhesion of its walls, but from a hollow epithelial sprout springing from that section from which the utricle comes. For each canal there are developed two sprouts which grow toward each other and finally unite, the cells of the outer (convex) side being larger than those of the inner. We may explain in this manner those cases of arrested development in which single canals have a blind ending.

15. His wishes to show the relation of single branches of the auditory nerve to certain portions of the future site of the ganglia. This is especially important as regards the middle branch of the auditory nerve, which supplies the sacculus rotundus and the posterior ampulla, and of which it has not been definitely known hitherto whether it formed a part of the cochlear or of the vestibular nerve. In the 3½–4-weeks-old embryo the site of the ganglia is composed of an unorganized complex of cells, which may, however, be divided into several parts: (1) the medial portion with the lateral root of the auditory nerve; (2) the lateral portion with the medial root of the auditory nerve; (3) the ventral portion in connection with the facial nerve. No trunk of the auditory nerve can be recognized, but the site of the ganglia lies close to the anterior wall of the aural vesicle. *From this anterior wall are developed all those portions of the labyrinth which later have nerve endings.* The posterior ampulla is an exception. It gets its innervation only at a later stage. The medial ganglion is in relation with the cochlea and the sacculus rotundus, the lateral ganglion on the contrary with the recessus utriculi, the anterior and external ampullæ. (His contradicts the statement of Rüdinger that the semicircular canals develop as hollow canals which unite, and is of the opinion that they arise through the growing together of folds.) At about the sixth week may be seen the middle branch of the auditory nerve, which arises from the central ganglion. The latter consists of ganglionic masses which in the development of the part are left in the immediate neighborhood of the cochlear canals. About the end of the second month the ear as a whole passes from the embryonal to the fœtal form. In the further development the central ganglion becomes

separated from the cochlear ganglion. The author has not come to a definite opinion as to the relation of the portio intermedia Wrisbergii to the acustico-facialis system, nor as to the intracerebral course of the roots of the auditory nerve. In conclusion he describes the modelling process of Selenka, by means of which the ear at different periods in its development may be reconstructed plastically from the sections.

16. HABERMANN reports a number of cases of disease of the internal ear in which there was a more or less complete destruction of the auditory nerve. The first case was a patient aged fifty-seven, who at the time of admission to the hospital was already so apathetic that no test of the hearing could be made. There was a depression in the parietal bone of eight years' standing. The patient was said to be deaf. At the autopsy were found, besides other pathological conditions, emphysema of the lungs, tuberculosis, Bright's disease, and marasmus. The petrous portion of the right temporal bone was removed and carefully studied. The middle ear showed evidences of previous purulent inflammation: hemorrhages and fatty degeneration of the tensor tympani and stapedius muscles. In the internal ear the nerve was wanting in the canalis ganglionaris, the lamina spiralis, and in a portion of Corti's organ. The nerve-sheath contained only a scanty mucoid connective tissue. In the lower turn the nerve was smaller than it should be in the adult. Besides this there were a number of hemorrhages, and at various points increase of pigment and amyloid bodies. The brain was not examined. The author supposes that the destruction of the auditory nerve was due to the wound of the cranium, and that the patient had but slight perception of tones of low pitch. The second case was a deaf-and-dumb woman, aged fifty, who had died of carcinoma of the stomach and liver. Here also only the petrous portion of the right temporal bone could be examined. In the middle ear there were evidences of fetid purulent inflammation. The epidermis had proliferated through the perforation to the promontory and the niche of the fenestra ovalis. There were a number of small cysts. There were hemorrhages between the nerve bundles, which were smaller than usual. In the basal turn the nerves were completely wanting in the lamina spinalis. The space usually occupied by the nerve-fibres was narrowed and filled with scanty mucoid connective-tissue and pigment cells. The canalis ganglionaris contained but few ganglion cells, which lay for the

most part in the half toward the internal auditory canal. Only vestiges of Corti's organ remained, or bits of pavement epithelium in its place. In the neighborhood of the fenestra rotunda there were still band-like masses, and near the membrane reticular connective tissue and new-formed bone. In the middle cochlear turn the changes were less marked. The changes were also less marked in the vestibule. Habermann believes the other ear to have been similarly diseased, and that the inflammatory process proceeded from the subarachnoidean space into the perilymphatic space of the internal ear. A continuation will follow.

17. GRANDENIGO studied three cases of tubercular meningitis, one case of purulent meningitis following severe inflammation of the middle ear, two cases of cerebro-spinal meningitis, and three cases of brain tumor. In the different forms of cerebral inflammation, inflammatory changes often occur in the trunk of the seventh or eighth cerebral nerves, the infection being carried through the internal auditory canal. The changes occur in the beginning more in the sheaths of the nerves than in the nerve substance. This is the case with the seventh nerve, but with the auditory nerve the pus presses in between the fibres as they divide to enter the modiolus, and causes their atrophy and destruction. In the majority of cases the disease does not pass the tabula cribrosa. In a few cases it extends to the spiral ganglion of Rosenthal in the lower turn. The author believes this to be the common passage for the extension of disease from the brain to the internal ear, the aquæductus cochleæ being always found intact. In most cases there were found inflammatory changes also in the middle ear, at least on one side, while no direct connection with the cerebral inflammation could be detected. Increased cerebral tension, which produces choked disc, may also cause a lymph stasis in the auditory nerve, which can be diagnosed only by the increased electric excitability.

18. SCHULTZE and KÜTTNER examined the organ of hearing and the spinal cord of a tubercular patient, who five years before became completely deaf from a disease beginning with cerebral symptoms. All the collections of ganglion cells considered as constituting the nucleus of the auditory nerve were found intact; the middle ear and the trunk of the facial nerve were also normal. Both auditory nerves were markedly atrophic. There was nothing to be seen of Corti's organ. The cavities of the internal ear were filled for the most part with osteoid tissue and round cells.

Schultze supposes that the process—cerebro-spinal meningitis or acute polimyelitis—extended into the internal ear through the nerve-trunks.

19. BAGINSKY compares the views of other investigators, especially Flechsig, Monakow, and Forel, on the central course of the auditory nerve in the rabbit, with his own, and describes as new, preparations from a cat, where eight weeks after destroying the cochlea he found a central atrophy. The posterior root of the nerve and the ganglion cells contained in it were completely atrophic. The tuberculum laterale was shrunken on the operated side, especially in its second and third layers. The anterior nucleus of the nerve was completely atrophic ; the external and internal nuclei showed no changes. The corpus trapezoides was moderately atrophic on the operated side, and the upper olivary body and the striæ medullares as well. On the side opposite that operated, the atrophy affected the lower lemniscus and the posterior corpus quadrigeminum. The author concluded from this : that the medial portion of the posterior root of the auditory nerve ends in the anterior nucleus ; the smaller lateral portion of the posterior root runs into the tuberculum laterale, the large medial into the anterior nucleus. In intimate relation with these two latter portions of the brain, is the corpus trapezoides in which the crossing of the posterior roots occurs.

21. MONAKOW cut through the lower lemniscus of a new-born dog. Other wounds were made unintentionally. The animal lived six weeks. The descending degeneration of the fibres could be traced in the dorsal substance of the upper right olivary body through the arciform fibres of the formatio reticularis and into the left striæ acusticæ. The left tuberculum acusticum showed atrophy. The right upper olivary body was moderately atrophic. The author, as before, considers the striated portion of the lower lemniscus as being in the secondary tract of the auditory nerve ; the elements of origin of this tract are the long ganglion cells in the tuberculum acusticum. The corpus trapezoides has nothing to do with this tract, it stands in no way in direct continuity with the lower lemniscus, and the author believes the opinions of Flechsig, Bechterew, and Baginsky on this subject to be incorrect.

b.—NASO-PHARYNX.

1. WINDLE, BERTRAM A. C., Birmingham. Three cases of malformation connected with the face. With five figures. *Anat. Anzeiger*, 1889, p. 219.

2. HERZFELD, J., Berlin. A contribution to the anatomy of the erectile tissue of the nasal mucous membrane. With one plate. *Arch. f. Micros., Anat.*, vol. xxxiv., p. 197, 1889.

3. DISSE, J., Berlin, formerly Tokio. The development of the nasal cavity after birth. With one plate. *Arch. f. Anat. u. Physiol.*, anat. Abth., Suppl. Band, p. 29, 1889.

4. BOTEY. On the structure of mucous polypi of the nasal fossæ. *Revue de laryng., d'otol.*, etc., vol. i., 1889.

5. PUTELLI, FERRUCCIO, Vienna. On the cells of the olfactory mucous membrane in chicken embryos at early stages. *Contributions from the University Embryological Institute*, Vienna, 1889, p. 26.

6. GRASSI, BATTISTA, and COSTIONOVO, A., Catania. A contribution to the knowledge of the olfactory organ of the dog. With one plate. *Arch. f. Microsc. Anat.*, vol. xxxiv., p. 385, 1889.

7. ALLEN, H. The anatomy of the nasal chambers. *New York Med. Journal*, 1889.

8. ONODI. An unusual case of congenital closure of the posterior nares. *Berliner klin. Wochenschr.*, No. 33, 1889.

9. OTTO, R., Dorpat. On a congenital hairy polypus of the pharynx. *Virchow's Arch.*, vol. cxv., p. 242.

10. ZAWARKIN, TH., St. Petersburg. On the epithelium of the tonsils. With two figures. *Anat. Anzeiger*, 1889, p. 467.

11. KOSTANECKI, KASIMIR. On the pharyngeal diverticulum in man, and the formation of diverticula in the naso-pharynx. With two plates from the Anatomical Institute in Berlin. *Virchow's Arch.*, vol. cxvii., p. 108.

12. PÖLCHEN, R. On the anatomy of the naso-pharynx. Read at a meeting of the Medical Society of Dantzic, Oct. 18th.

13. POTIQUET. The pharyngeal or Luschka's bursa. Anatomical studies. *Revue de laryng., d'otol.*, etc., 1889, No. 24.

14. TROLARD. On the central nervous olfactory apparatus. *C. R. Soc. de Biol.*, 1889, p. 664.

1. The first case was a rare and, in many respects, interesting medial cleft of the nose; the second an unusual form of cleft palate. The third case described was one of rudimentary development (almost defect) of the right eye, having, at the same time, appendices before both auricles.

2. *a.* The erectile tissue of the nasal mucous membrane is rich in organic muscle. This does not lie free in the areolar tissue of

the erectile body, but, as in other situations, forms the tunica media of the arteries and veins, and, further, lies closely packed about the lacunæ of the erectile body.

b. In the areolar tissue of the erectile body there are a number of elastic fibres.

c. The turbinate bone is spongy, containing marrow. The erectile tissue does not extend into the bone, but veins come from the bone and anastomose with the veins of the periosteum and the outlet channels of the erectile tissue.

3. The material studied comprised the skulls of twenty-six children and twelve adults. The point chiefly considered in the investigations was the question why it is that difficulty in breathing—and, in consequence of this, threatening symptoms from the want of air—occur so often, under certain conditions, in children and so rarely in adults. The results were as follows : The structure of the nasal cavity in infants and children differs considerably from that in adults. The cavity is low in the new-born ; it develops downward, and thus the olfactory region is removed from the floor of the cavity. The respiratory region increases in breadth and height. While the whole nasal cavity increases two and one half times in size, the respiratory region alone increases three times in its growth from birth up to the state of complete development. The increase in width during the same period is still more considerable.

6. Preparations made by a very difficult method of staining, show that the varicose fibres of the olfactory nerves arriving in the connective tissue of the conjunctiva or at the boundary between connective tissue and epithelium, divide in such manner that their twigs take a course almost horizontal, and these latter send off twigs which, running into the epithelium, end in the olfactory cells. Single fibres may run directly to the olfactory cells without taking a horizontal course. Nowhere are there evidences of anastomosis.

10. ZAWARKIN describes, in perpendicular sections of the tonsil of a dog, peculiar flask-shaped spaces situated in or below the epithelium, which are filled with leucocytes and open on the surface by comparatively narrow ducts.

9. A polypus of hazel-nut size was removed from the pharynx of a seventeen-hour old child, which, especially when placed on its back, suffered from severe dyspnoic symptoms. The polypus, in its microscopic structure, was similar to the hairy skin, lacking

only the papillæ. In its centre was a quantity of fat and a small plate of cartilage. As to its morphology, the author inclines toward the opinion of Arnold that it represented an undeveloped twin ovum.

11. In the region of the first pharyngeal arch, KOSTANECKI explains the recessus salpingo-pharyngeus of Zuckerkandl, and similar formations, by supposing a congenital dilatation of the membranous portion at the lower part of the cartilaginous tube, produced by the whole configuration of the naso-pharyngeal cavity. The diverticulum in the tube described by Kirschner was observed a number of times by Kostanecki, and he explains it as a dilatation of the tube wall produced in the embryo mechanically by pressure in the lumen of the tube (retained secretion). The diverticula of the second pharyngeal arch are considered merely extraordinary forms of Rosenmüller's fossa.

12. PÖLCHEN examined preparations from the roof of the pharynx of a large number of adults, and found that there is a spot in the middle of the naso-pharynx which, in comparison with its surroundings, possesses anatomical peculiarities. Tornwaldt was right when he thought the so-called bursa pharyngea to be a depression in the mucous membrane, and was right when he considered this point to be the most frequent and important site of pathological secretion.

13. POTIQUET examined 16 skulls of individuals from 9 to 70 years of age. In every case he found the tonsil of the pharynx more or less preserved. As to the question whether the bursa described by Luschka be anatomically an individual structure, he stated that he had found but twice a depression (2 and 4 *mm*) near the posterior margin of the space originally occupied by the pharyngeal tonsil. He supposes this to be produced by the atrophy of the tonsil. The name hitherto given to this depression seems to him unsuitable. He approves Robin's term, "foramen cœcum." (The literature has not been sufficiently examined; the preparations are in part unadapted, in part too superficially studied, to add any thing new to our existing knowledge.—REV.).

II.—PHYSIOLOGY AND PHYSICS.

a.—EAR.

1. DOWLING, F. On the auricle and its physiological significance. *Lancet Clinic*, May 18, 1889.

2. RICHEY, S. O. The physiology of the intra-tympanic muscles. *Jour. of the Amer. Med. Ass'n*, vol. xiii., p. 249.

3. STEFANINI, A. On the least energy capable of producing the sensation of sound. *Il nuovo Cimento*, ser. iii., vol. xxiv., p. 215.

4. LOVE, J. KERR. The limits of audition. *Jour. of Anat. and Physiol.*, 1889, vol. xxiii., p. 336.

5. PREYER, W. On combinational tones. *Ann. d. Phys. u. Chemie.*, vol. xxxviii., p. 131.

6. GRADENIGO, GIUSEPPE, Turin. On the electric reaction of the nervus acusticus and its graphic representation. *Arch. f. Ohrenheilk.*, vol. xxviii., pp. 191 and 241.

7. GELLÉ. On hearing in a noise. A critical experimental study. *Revue de laryng., d'otol.*, etc., 1889, p. 341.

8. LICHTWITZ, R., Bordeaux. On the use of the new Edison phonograph as a general instrument for determining the power of hearing. *Prager. med. Wochenschr.*, 1889, No. 47.

9. LICHTWITZ, R. A note on the same subject, presented by Janssen. *Compt. rend.*, 1889, p. 473.

10. DENNERT, H., Berlin. Acoustico-physiological investigations and studies adapted to practical otology. *Arch. f. Ohrenheilk.*, vol. xxix., p. 68.

11. KÖNIG, RUDOLPH, Paris. On the phenomena resulting from the combination of two tones and one tone-color (timbre). *Tagebl. d. 62. Vers. deutscher. Naturf. u. Aerzte*, in Heidelberg, 1889, p. 199.

12. STEFANINI, A. On the laws of oscillation of the diapason and on the measurement of the intensity of sound. *Il nuovo Cimento*, vol. xxvi., pp. 157 and 193.

13. GREGORY, G. On a method of driving tuning-forks electrically. *Philos. Mag. and Jour. of Science*, vol. xxviii., p. 490.

14. TUMA. Observations of the vibration of tuning-forks with the aid of the microphone. *Sitzungsber. d. k. Akad d. W. in Wien*, vol. xcviii., Abth. ii., a, p. 1028.

15. DREHER, E. The physiology of music. Halle, 1889.

16. DELAGE, Yves. The non-auditory function of the internal ear. *Rev. Scientif.*, 1889, vol. ii., p. 610.

17. EWALD, J. R. On motor disturbances after injury to the semicircular canals. *Centralbl. f. d. med. Wissensch.*, 7, 8.

18. MERCADIER, M. E. (note by). On telephonography. *Compt. rend.*, vol. cviii., p. 670.

3. Wead, in the year 1883, endeavored to find by experiment the slightest energy which, per second and square centimetre, is capable of causing an appreciable stimulation to the ear. His experiments showed that the tones of the middle octave are those most readily perceived by the ear. Alluard's experiments in the year 1882 seemed to show that the energy required is less for low-pitched tones than for acute. STEFANINI subjects Wead's measurements to a theoretical examination, and comes to the conclusion that, independent of the intensity of the tone, within experimental limits the sensibility of the ear is the same for tones of every pitch. (This question, however, is not settled by this paper.—REV.)

4. The author considers : (1) The lowest pitched note perceptible ; (2) the highest pitched note perceptible ; (3) the appreciation of small differences in pitch ; (4) the distance at which a tone of given intensity can be heard ; (5) tone or note deafness. His individual experiments cover only the third point ; they were made by means of changeable pipes. His conclusions were : It is hard to determine the smallest difference in pitch which is appreciated by the uncultivated ear ; it varies between $\frac{1}{6}$ and $\frac{1}{40}$ of a semitone. The average difference may be $\frac{1}{24}$ of a semitone. The ear of a cultivated musician will appreciate with certainty a difference of from $\frac{1}{64}$ to $\frac{1}{80}$ of a semitone. All observers discover more readily a difference in tone when the second note is of higher pitch than when it is of lower. In general, Weber's law holds, excepting the highest and lowest tones, for all portions of the musical scale.

5. Former experiments seemed to indicate that it was impossible to prove the existence of differential and compound tones even with the assistance of the most sensitive tuning-forks and resonators. PREYER found, on the contrary, that with a tuning-fork of $\mp$ 2 vibrations difference applied to the ear, the differential tone of the first order will still send vibrations to the ear when the two objective tones can no longer be heard ; and that a resonator having its number of vibrations equal to that of the differential tone, if previously applied to the ear, will give beats with such a fork held in the air. The same air in the resonator which, when held away from the ear, is not brought into harmony with the differential tone, is brought into similar vibration with it

when applied to the ear, in case the differential tone be audible. PREYER shows further that combinational tones have their origin in the membrana tympani and not in the internal ear; when the ear is closed (bone-conduction), or when there is perforation of the membrana tympani, they are not heard. If, however, a small perforation be closed with a drop of water the differential tones are at once perceived. Compound tones, contrary to the former views in accord with the theory of Helmholtz, were shown to be produced by the objective addition of the vibrations of two tones.

6. After extensive investigations GRADENIGO makes the following statements: (1) On account of the frequency of diseases of the ear and of the nervous system, in the study of the reaction of the auditory nerves to electric stimulation, we cannot take into consideration all those observations which were formerly made on persons whom careful functional and objective examination showed to be healthy. (2) With persons really healthy it is possible to obtain an electric reaction of the auditory nerves to a degree varying from 5–12 per cent. (3) In healthy persons with whom it is possible to get a reaction, generally a current of from 10–16 *m.-a.*, always a current stronger than 6 *m.-a.*, is required at the first sitting, while with subjects having disease of the ear or nervous system a more or less complete reaction is obtained generally with a current of from 1–3 *m.-a.*, always by a current weaker than 6 *m.-a.* (4) The fact that it is possible with a healthy subject to cause a reaction of the auditory nerve by electric stimulation externally must be regarded as a characteristic clinical peculiarity in that person. From a new series of studies of patients the conclusion is arrived at: That in the great majority of cases, in order to explain the slight electric reaction of the auditory nerve, we must assume an increase of sensibility to stimulation in the nerve; whether in other cases (purulent inflammation of the middle ear) there be another factor besides this is not clearly proven. It is probable that the physical condition of the auditory canal, the membrana tympani, and the cavity of the tympanum, has little influence on the electric reaction of the auditory nerve when the external method is used. According to other observations it is at least doubtful whether hyperæmia and exudation in the middle ear lessen to any considerable degree the electric resistance. It is very probable that the so-called electric reaction of the auditory nerve should be referred not to the

terminal apparatus (acoustic cells), but rather to the nerve trunk itself and its peripheral branches. With a unilateral purulent inflammation of the middle ear, the sensibility to electric stimulation may be increased in the nerve of the healthy side. An appendix follows the paper, in which graphic representations are given of the reaction in its various forms. The strength of current employed, the pitch and the duration of the sound perceived, are represented by colors in quadratic fields, offering thus a picture easily understood.

7. GELLÉ, from experiments on his own person, concludes that with the occurrence of a second physical or nervous stimulation, the sensibility of perception of the ear is increased for acoustic stimuli.

8 and 9. As Edison's representative passed through Europe last year with the phonograph, he stated that the instrument was adapted to a number of purposes, among others to the testing of hearing. A practical test was never made however. LICHTWITZ has now studied the matter from a theoretic standpoint. Whether the phonograph is adapted to this purpose must however be doubted until practical investigations are made regarding it.

10. DENNERT, after experiments of his own and consideration of well-known phenomena, comes to the conclusion that we are wrong in supposing noise and musical sound to be for the ear two different vibratory phenomena. He believes that the cochlea suffices for the perception of both. He suggests, in addition to the hearing tests previously in use, that of the perception of tones of very short duration. He makes the test either by means of vibrating forks carried quickly past the ear, or, better, by means of resonators struck by a small hammer whose falling distance is variable. It is not improbable that this new method of examination may become a useful member of the series of hearing tests.

11. When two tones, with vibrations in directions at right angles to each other, are produced in one body, loud beats will be heard exactly as if the two tones had been produced by two distinct sources. The tone-color is not caused by harmonics alone, since the secondary tones of a vibrating body never correspond exactly to their theoretic values. Therefore tones are often, and with some instruments always, accompanied by secondary tones not purely harmonic, having waves of an ever-changing form. There are also sounds produced by waves of a constant length

and breadth but of different forms, and this change of form is quite arbitrary, but not caused by impure harmonics. Corresponding tone-tracings are given in the paper and experiments with the syren discs described.

b.—NASO-PHARYNX.

1. LÖWENBERG, Paris. Acoustic studies on the nasal sounds. *Deutsche med. Wochenschr.*, 1889, No. 26.

2. ZWAARDEMAKER, H. Cocaine anæmia. *Fortschr. d. Med.*, vol. vii., p. 481.

3. ZWAARDEMAKER, H. Compensation of odors by means of the double olfactometer. *Fortschr. d. Med.*, vol. vii., p. 721.

1. LÖWENBERG studied the physical difference between the French nasal sounds (*an*, *ein*, *on*) and those not French (*ang*, *eng*, *ong*). He produced the sound desired, left the cavities employed in its production in the same position, and by means of a graded series of König's tuning-forks found what tone gave the strongest resonance. It was found that the primary tone of the French sound was equal to the lower third of the primary tone of the corresponding pure sound, while the corresponding sound not French was about a semitone higher than the lower octave of the corresponding pure sound. (The statement of the propositions as well as the method of the examinations is not altogether above criticism.—REV.)

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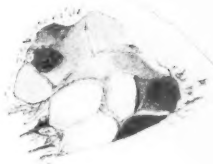
I

II

III



IV

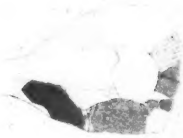
V

VI

VII

VIII

IX



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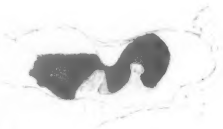

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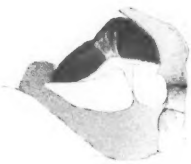

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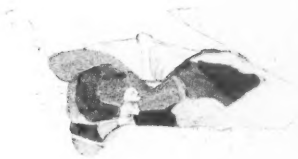

XIII

XIV

XV

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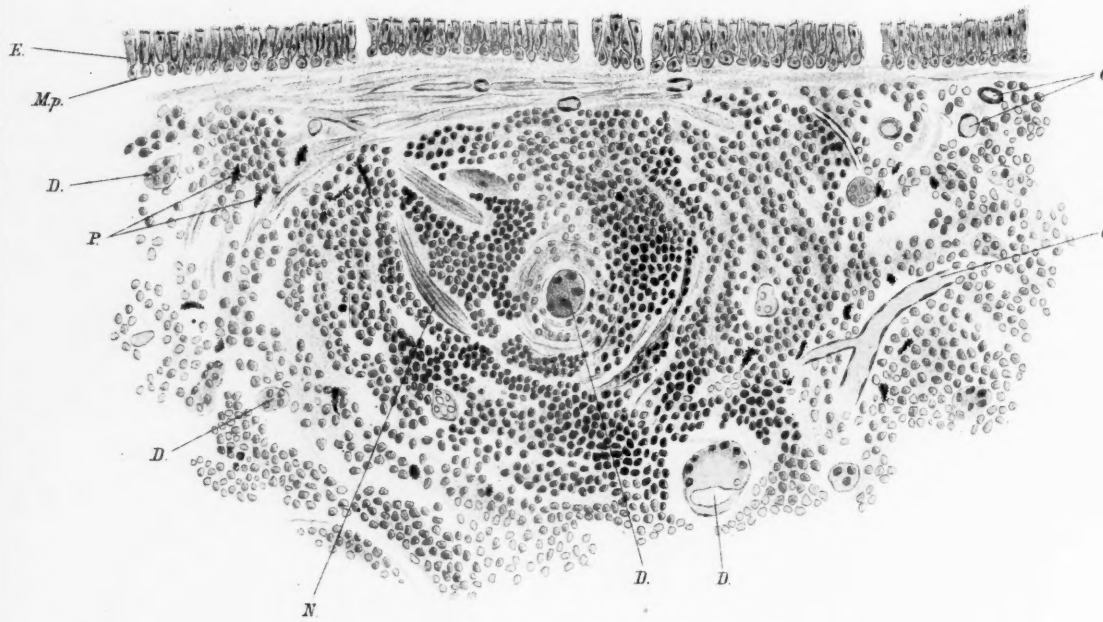
Fig. I.

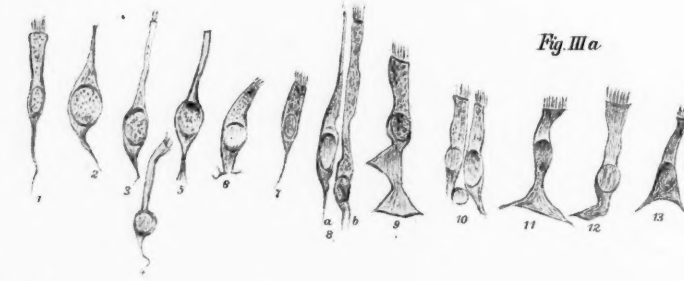


Olfactory region (left upper turbinated bone) in a case of Leukaemia (Leitz Ocular 3, Hartnack Object 4).

E = Epithelium. N = Nerve.
Mp = Membrana propria. G = Blood vessels.
D = Remains of Bowmans glands. P = Pigment.

The greater part of the specimen consists of adenoid tissue with extensive infiltration of leucocytes.

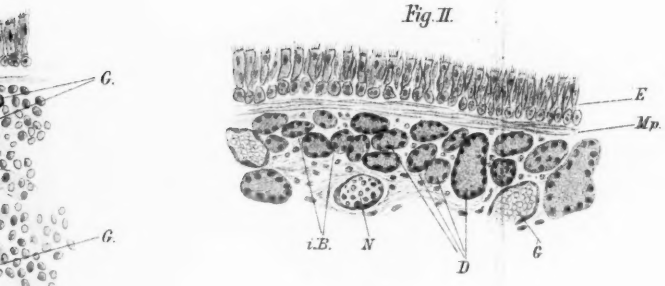
Fig. III a



2, 3, 5, 7, 8a Cells of smelling
1, 9, 10, 11, 12, 13 Supporting cells.
16, 19 Deep leying cells.
17 Substituting cells.
15 Supporting cell; the upper part degenerated in to mucus which the cell is about to discharge; the nucleus is plainly visible near the base of the cell.

1–13 Cells fr
part of t
24–31 do. bu
septum
ted bon
14–23 Cells
the upp
turbinat
23a–23e Cells
left mid

Fig. II.



Olfactory region from a man 40 yrs old with normal seuse of smell.
E = Epithelium.
Mp = Membrana propria.
D = Bowman's glands.
N = Nerve.
G = Blood vessels.
i. B = interglandulas tissue.

Fig. III b.

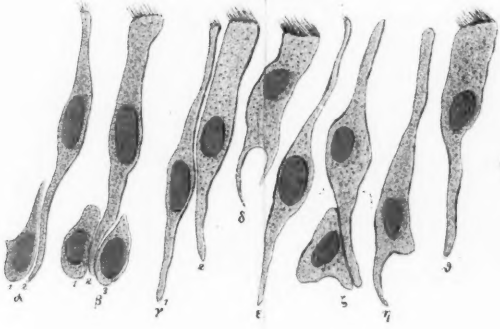
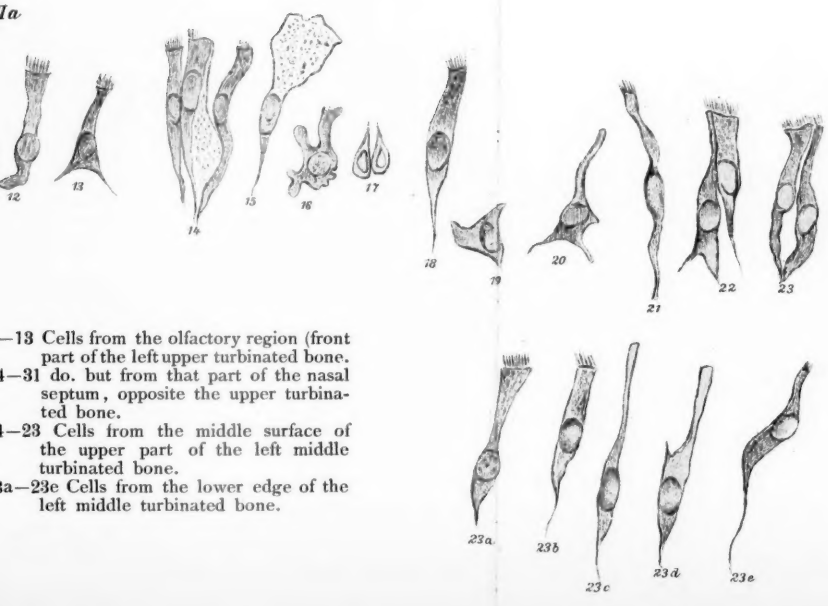


Fig. III bb

Size of some of the ciliated cells.
Lagest { 0,075 long, nucleus 0,006 broad, 0,015 long, 0,06 Mill. long; upper end 0,018 Mill. long,
Middle size 0,042—0,045 Mill. long.
Smollest 0,024—0,027 Mill. long.

1a



—13 Cells from the olfactory region (front part of the left upper turbinated bone.
4—31 do. but from that part of the nasal septum, opposite the upper turbinated bone.
4—23 Cells from the middle surface of the upper part of the left middle turbinated bone.
3a—23e Cells from the lower edge of the left middle turbinated bone.

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